```
Trying 3106016892...Open

Welcome to STN International!  Enter x:x
LOGINID:ssspta1617srh
PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2


* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1          Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2  Dec 17  The CA Lexicon available in the CAPLUS and CA files
 NEWS  3  Feb 06  Engineering Information Encompass files have new names
 NEWS  4  Feb 16  TOXLINE no longer being updated
 NEWS  5  Apr 23  Search Derwent WPINDEX by chemical structure
 NEWS  6  Apr 23  PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
 NEWS  7  May 07  DGENE Reload

 NEWS EXPRESS  April 18 CURRENT WINDOWS VERSION IS V6.0,
               CURRENT MACINTOSH VERSION IS V5.0C (ENG) AND V5.0JB (JP),
               AND CURRENT DISCOVER FILE IS DATED 04/06
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
 NEWS LOGIN    Welcome Banner and News Items
 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that
specific topic.

  All use of STN is subject to the provisions of the STN Customer
  agreement.  Please note that this agreement limits use to scientific
  research.  Use for software development or design or implementation
  of commercial gateways or other similar uses is prohibited and may
  result in loss of user privileges and other penalties.

  * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * *



FILE 'HOME' ENTERED AT 11:31:54 ON 14 MAY 2001

=> fil reg
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.45       0.45

FILE 'REGISTRY' ENTERED AT 11:33:36 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 American Chemical Society (ACS)

STRUCTURE FILE UPDATES:   13 MAY 2001  HIGHEST RN 335276-85-2
DICTIONARY FILE UPDATES:  13 MAY 2001  HIGHEST RN 335276-85-2

TSCA INFORMATION NOW CURRENT THROUGH January 11, 2001

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.
```

Structure search limits have been increased. See HELP SLIMIT
for details.

=> s misoprostol/cn
L1 1 MISOPROSTOL/CN

=> fil stng
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 4.11 4.56

FILE 'STNGUIDE' ENTERED AT 11:34:08 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May 11, 2001 (20010511/UP).

=> file hcaplus
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 0.00 4.56

FILE 'HCAPLUS' ENTERED AT 11:35:06 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1947 - 14 May 2001 VOL 134 ISS 21
FILE LAST UPDATED: 13 May 2001 (20010513/ED)

This file contains CAS Registry Numbers for easy and accurate
substance identification.

This file supports REG1stRY for direct browsing and searching of
all substance data from the REGISTRY file. Enter HELP FIRST for
more information.

HCAplus now provides online access to patents and literature covered
in CA from 1947 to the present. On April 22, 2001, bibliographic
information and abstracts were added for over 2.2 million references
published in CA from 1947 to 1966.

=> s misoprostol or l1
845 MISOPROSTOL
727 L1
L2 882 MISOPROSTOL OR L1

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (a)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia)

```
UNMATCHED LEFT PARENTHESIS 'S) ((SEX?'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (a)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia))
        103473 FEMALE
         47254 FEMALES
        130450 FEMALE
                 (FEMALE OR FEMALES)
          4603 WOMAN
             9 WOMANS
         58044 WOMEN
            23 WOMENS
         60685 WOMAN
                 (WOMAN OR WOMANS OR WOMEN OR WOMENS)
         58044 WOMEN
            23 WOMENS
         58053 WOMEN
                 (WOMEN OR WOMENS)
          1029 GIRL
          2705 GIRLS
          3554 GIRL
                 (GIRL OR GIRLS)
           328 LADY
           132 LADIES
           457 LADY
                 (LADY OR LADIES)
        108098 SEX?
          5157 GENITAL
           154 GENITALS
          5267 GENITAL
                 (GENITAL OR GENITALS)
           792 HYPOACTIV?
        123498 DESIR?
        138810 SATISFACT?
            51 ORGASM
          3156 AROUSAL?
          2173 SENSATION?
       1553151 RESPON?
           121 ANHEDONIA
L3         145 (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENITAL
               ) (A) (HYPOACTIV? OR DESIR? OR SATISFACT? OR ORGASM OR
AROUSAL?
               OR SENSATION? OR RESPON? OR ANHEDONIA))

=> s scan
         19854 SCAN
          9143 SCANS
L4        27636 SCAN
                 (SCAN OR SCANS)

=> d scan

L4    27636 ANSWERS   HCAPLUS  COPYRIGHT 2001 ACS
IC   ICM  H01L021-66
     ICS  H01L021-66; G01B015-00; G01B015-04; G01N001-28; G01N001-32;
          G01N023-04; G01N023-225; H01J037-22; H01J037-26; H01L021-3065
```

TI Defective inspection + defective method for analyzing of semiconductordevice pattern, survey instrument of defective inspection + failure analysis system, and semiconductor device pattern of semiconductor device pattern. [Machine Translation].

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):2

L4 27636 ANSWERS HCAPLUS COPYRIGHT 2001 ACS
IC ICM G02F001-1343
ICS G02F001-1337; G09F009-30; G09F009-35
TI Liquid crystal display. [Machine Translation].

L4 27636 ANSWERS HCAPLUS COPYRIGHT 2001 ACS
IC ICM G01R031-02
ICS H05K003-00
TI Etched circuit substrate inspection vessel. [Machine Translation].

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L4 27636 ANSWERS HCAPLUS COPYRIGHT 2001 ACS
IC ICM G01M011-00
ICS G02F001-13
TI Grade inspection method and its grade survey instrument of plane surface display. [Machine Translation].

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> s ((sex? or genital) (a) (hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or respon? or anhedonia))
108098 SEX?
5157 GENITAL
154 GENITALS
5267 GENITAL
(GENITAL OR GENITALS)
792 HYPOACTIV?
123498 DESIR?
138810 SATISFACT?
51 ORGASM
3156 AROUSAL?
2173 SENSATION?
1553151 RESPON?
121 ANHEDONIA
L5 595 ((SEX? OR GENITAL) (A) (HYPOACTIV? OR DESIR? OR SATISFACT? OR ORGASM OR AROUSAL? OR SENSATION? OR RESPON? OR ANHEDONIA))

=> s ((sex? or genital) (s) (hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or respon? or anhedonia))
108098 SEX?
5157 GENITAL
154 GENITALS
5267 GENITAL
(GENITAL OR GENITALS)
792 HYPOACTIV?
123498 DESIR?
138810 SATISFACT?
51 ORGASM
3156 AROUSAL?
2173 SENSATION?
1553151 RESPON?

```
          121 ANHEDONIA
L6       12767 ((SEX? OR GENITAL) (S) (HYPOACTIV? OR DESIR? OR SATISFACT? OR
               ORGASM OR AROUSAL? OR SENSATION? OR RESPON? OR ANHEDONIA))

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia)
UNMATCHED LEFT PARENTHESIS 'S) ((SEX?'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia) )
        103473 FEMALE
         47254 FEMALES
        130450 FEMALE
                 (FEMALE OR FEMALES)
          4603 WOMAN
             9 WOMANS
         58044 WOMEN
            23 WOMENS
         60685 WOMAN
                 (WOMAN OR WOMANS OR WOMEN OR WOMENS)
         58044 WOMEN
            23 WOMENS
         58053 WOMEN
                 (WOMEN OR WOMENS)
          1029 GIRL
          2705 GIRLS
          3554 GIRL
                 (GIRL OR GIRLS)
           328 LADY
           132 LADIES
           457 LADY
                 (LADY OR LADIES)
        108098 SEX?
          5157 GENITAL
           154 GENITALS
          5267 GENITAL
                 (GENITAL OR GENITALS)
           792 HYPOACTIV?
        123498 DESIR?
        138810 SATISFACT?
            51 ORGASM
          3156 AROUSAL?
          2173 SENSATION?
       1553151 RESPON?
           121 ANHEDONIA
L7        1933 (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENITAL
               ) (S) (HYPOACTIV? OR DESIR? OR SATISFACT? OR ORGASM OR
AROUSAL?
               OR SENSATION? OR RESPON? OR ANHEDONIA) )

=> s l2 and l7
L8           2 L2 AND L7

=> d ti tot
```

L8 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2001 ACS
TI Use of **misoprostol** or/and **misoprostol** acid for preparing drug in order to cure sexual dysfunction in women

L8 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2001 ACS
TI Methods, compositions, and kits for enhancing **female sexual desire** and **responsiveness** using prostaglandins

=> d ibib abs 2

L8 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:282087 HCAPLUS
DOCUMENT NUMBER: 130:321230
TITLE: Methods, compositions, and kits for enhancing **female sexual desire** and **responsiveness** using prostaglandins
INVENTOR(S): Neal, Gary W.
PATENT ASSIGNEE(S): Androsolutions, Inc., USA
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9920266	A1	19990429	WO 1998-US21631	19981020
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9896952	A1	19990510	AU 1998-96952	19981020
EP 1028720	A1	20000823	EP 1998-951063	19981020
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1997-954122 A 19971020
WO 1998-US21631 W 19981020

AB Topical application of a prostaglandin directly to the clitoris is effective for enhancing **female sexual desire** and **responsiveness**. Kits and pharmaceutical compns. contg. the prostaglandins are claimed as well. The pharmaceutical compns. may contain at least one coagent as well selected from the group consisting of 15-hydroxyprostaglandin dehydrogenase inhibitors, ACE inhibitors, nitro vasodilators, alpha blockers, yohimbine, labetalol, carvedilol, bucindolol, phosphodiesterase inhibitors, muscarinic agents, dopaminergic agonists, ergot alkaloids, opiate antagonists, and polypeptide neurotransmitters.

REFERENCE COUNT: 2
REFERENCE(S): (1) Hadley; US 5576290 A 1996 HCAPLUS
(2) Roy, A; British Journal of Urology 1989, V64, P180

MEDLINE

```
=> fil stng
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                        26.66      31.22

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)              SINCE FILE      TOTAL
                                                           ENTRY    SESSION
CA SUBSCRIBER PRICE                                        -0.59      -0.59

FILE 'STNGUIDE' ENTERED AT 11:42:34 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May 11, 2001 (20010511/UP).

=> file medline caplus embase biosis uspatfull
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.00      31.22

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)              SINCE FILE      TOTAL
                                                           ENTRY    SESSION
CA SUBSCRIBER PRICE                                         0.00      -0.59

FILE 'MEDLINE' ENTERED AT 11:52:55 ON 14 MAY 2001

FILE 'CAPLUS' ENTERED AT 11:52:55 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'EMBASE' ENTERED AT 11:52:55 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V.  All rights reserved.

FILE 'BIOSIS' ENTERED AT 11:52:55 ON 14 MAY 2001
COPYRIGHT (C) 2001 BIOSIS(R)

FILE 'USPATFULL' ENTERED AT 11:52:55 ON 14 MAY 2001
CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

=> e neal, gary/au
E1            9     NEAL ZANE C/AU
E2            1     NEAL ZANE CLAY/AU
E3            0 --> NEAL, GARY/AU
E4            1     NEALAND S/AU
E5           18     NEALE A/AU
E6            4     NEALE A B/AU
E7            8     NEALE A C/AU
E8           18     NEALE A D/AU
E9            1     NEALE A E T/AU
E10          13     NEALE A J/AU
E11           1     NEALE A M/AU
E12           4     NEALE A R/AU

=> e neal gary/au
```

```
E13          2     NEAL GARDNER S/AU
E14          2     NEAL GARRISON R/AU
E15          0 --> NEAL GARY/AU
E16          1     NEAL GARY F/AU
E17          1     NEAL GARY R/AU
E18         10     NEAL GARY W/AU
E19          3     NEAL GENEVIEVE S/AU
E20          1     NEAL GEORGE/AU
E21          2     NEAL GEORGE F/AU
E22          1     NEAL GEORGE H/AU
E23          1     NEAL GERALD V/AU
E24          2     NEAL GERALD WAYNE/AU

=> e e18
E25          1     NEAL GARY F/AU
E26          1     NEAL GARY R/AU
E27         10 --> NEAL GARY W/AU
E28          3     NEAL GENEVIEVE S/AU
E29          1     NEAL GEORGE/AU
E30          2     NEAL GEORGE F/AU
E31          1     NEAL GEORGE H/AU
E32          1     NEAL GERALD V/AU
E33          2     NEAL GERALD WAYNE/AU
E34          1     NEAL GERTRUDE M/AU
E35          3     NEAL GORDON/AU
E36         62     NEAL GORDON E/AU

=> s e18
L9          10 "NEAL GARY W"/AU

=> d ti so tot

L9   ANSWER 1 OF 10  CAPLUS  COPYRIGHT 2001 ACS
TI   Methods and compositions for preventing and treating urinary tract
     disorders
SO   PCT Int. Appl., 32 pp.
     CODEN: PIXXD2

L9   ANSWER 2 OF 10  CAPLUS  COPYRIGHT 2001 ACS
TI   Prostaglandin derivatives for preventing and treating prostate disorders
SO   PCT Int. Appl., 40 pp.
     CODEN: PIXXD2

L9   ANSWER 3 OF 10  CAPLUS  COPYRIGHT 2001 ACS
TI   Methods, compositions, and kits for enhancing female sexual desire and
     responsiveness using prostaglandins
SO   PCT Int. Appl., 51 pp.
     CODEN: PIXXD2

L9   ANSWER 4 OF 10  CAPLUS  COPYRIGHT 2001 ACS
TI   male erectile dysfunction with a prostaglandin vasodilator and a
     15-hydroxyprostaglandin dehydrogenase inhibitor, and suppository
     composition
SO   PCT Int. Appl., 67 pp.
     CODEN: PIXXD2

L9   ANSWER 5 OF 10  CAPLUS  COPYRIGHT 2001 ACS
TI   Insulin degradation by human skeletal muscle
SO   Biochim. Biophys. Acta (1982), 719(2), 259-66
     CODEN: BBACAQ; ISSN: 0006-3002
```

L9 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2001 ACS
TI Catecholborane (1,3,2-benzodioxaborole). A versatile reducing agent
SO J. Org. Chem. (1977), 42(3), 512-17
CODEN: JOCEAH

L9 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2001 ACS
TI Organoboranes as alkylating agents
SO J. Chem. Educ. (1976), 53(9), 549-50
CODEN: JCEDA8

L9 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2001 BIOSIS
TI Methods for treating male erectile dysfunction.
SO Official Gazette of the United States Patent and Trademark Office Patents,
(Aug. 15, 2000) Vol. 1237, No. 3, pp. No Pagination. e-file.
ISSN: 0098-1133.

L9 ANSWER 9 OF 10 USPATFULL
TI Methods for treating male erectile dysfunction

L9 ANSWER 10 OF 10 USPATFULL
TI Method and means for linearizing a voltage controlled oscillator sweep generator

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s) (hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or respon? or anhedonia))
<------User Break------>
u
SEARCH ENDED BY USER
SEARCH ENDED BY USER

=> fil medline embase biosis uspatfull
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	13.43	44.65

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'MEDLINE' ENTERED AT 11:57:31 ON 14 MAY 2001

FILE 'EMBASE' ENTERED AT 11:57:31 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 11:57:31 ON 14 MAY 2001
COPYRIGHT (C) 2001 BIOSIS(R)

FILE 'USPATFULL' ENTERED AT 11:57:31 ON 14 MAY 2001
CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s) (hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or respon? or anhedonia))
3 FILES SEARCHED...
L10 43446 (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR GENITAL

) (S) (HYPOACTIV? OR DESIR? OR SATISFACT? OR ORGASM OR AROUSAL?
OR SENSATION? OR RESPON? OR ANHEDONIA))

=> s l1 or misoprostol
L11 6378 L1 OR MISOPROSTOL

=> s l10 and l11
L12 1 L10 AND L11

=> d ibib abs

L12 ANSWER 1 OF 1 USPATFULL
ACCESSION NUMBER: 1998:42085 USPATFULL
TITLE: Transdermal drug delivery device for treating erectile dysfunction
INVENTOR(S): Lee, Hun Han, Taejeon, Korea, Republic of
Cho, Joong Woong, Taejeon, Korea, Republic of
Kim, Choul Young, Taejeon, Korea, Republic of
Pai, Chaul Min, Taejeon, Korea, Republic of
Song, Jin Deog, Taejeon, Korea, Republic of
Park, Chul Min, Taejeon, Korea, Republic of
Yoon, Hye Jeong, Taejeon, Korea, Republic of
Yeo, Yoon, Taejeon, Korea, Republic of
Paick, Jae Seung, Seoul, Korea, Republic of
PATENT ASSIGNEE(S): Sam Yang Co., Ltd., Seoul, Korea, Republic of (non-U.S. corporation)

	NUMBER	DATE	
PATENT INFORMATION:	US 5741511	19980421	
APPLICATION INFO.:	US 1996-627805	19960410	(8)
PRIORITY INFORMATION:	KR 1995-8444	19950412	
	KR 1995-43422	19951124	
	KR 1996-6908	19960315	

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Phelan, D. Gabrielle
LEGAL REPRESENTATIVE: Kenyon & Kenyon
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 445

AB The present invention is to provide a method and a transdermal drug delivery device for treating erectile dysfunction which comprises a patch containing pharmaceutically active ingredient and being directly apply to the male glans penis and its support and the rings for constricting the base part of the penis to aid the erection. The patch according to the present invention may be divided into two types, i.e. a cylinder type patch and a multi-reservoir type patch. The transdermal drug delivery patch device of the present invention is painless and safely to use and may be effectively produced and maintained erection of the penis without the adverse side effects and with a high degree of patient acceptability in the case of male impotence.

=> d kwic

L12 ANSWER 1 OF 1 USPATFULL
SUMM . . . a condom may cause the side effects including allergy due to the condom to the male as well as the **female**, and may not give the **orgasm** during the **sexual** intercourse. Therefore, such patch system may not apply to the persons having the rejection symptoms on the use of condom.
DETD Ditiazem, nifedipine, nicardipine, nimodipine and/or verapamil can be used as calcium blockers. Further, PGE1, alprostadil, **misoprostol** and/or PGE2 as natural and synthetic vasoactive prostaglandins; apomorphine, bromocriptine as dopamine antagonists; adenosine, amylnitrate, dipyridamole, erythrityl tetranitrate, ethaverine hydrochloride,. . .

=> fil stng

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	5.72	50.37

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'STNGUIDE' ENTERED AT 12:00:21 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May 11, 2001 (20010511/UP).

=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.00	50.37

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, ...' ENTERED AT 12:02:52 ON 14 MAY 2001

59 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view search error messages that display as 0* with SET DETAIL OFF.

=> fil medline embase biosis uspatfull

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.45	50.82

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE ENTRY 0.00 TOTAL SESSION -0.59

CA SUBSCRIBER PRICE

FILE 'MEDLINE' ENTERED AT 12:03:09 ON 14 MAY 2001

FILE 'EMBASE' ENTERED AT 12:03:09 ON 14 MAY 2001

FILE 'BIOSIS' ENTERED AT 12:03:09 ON 14 MAY 2001

FILE 'USPATFULL' ENTERED AT 12:03:09 ON 14 MAY 2001

=> s l1 or misoprostol or (prostagladin E) or PGE
L13 23010 L1 OR MISOPROSTOL OR (PROSTAGLADIN E) OR PGE

=> s l13 and l10
L14 55 L13 AND L10

=> dup rem l14
PROCESSING COMPLETED FOR L14
L15 41 DUP REM L14 (14 DUPLICATES REMOVED)

=> s l13 (s) l10
L16 37 L13 (S) L10

=> dup rem l16
PROCESSING COMPLETED FOR L16
L17 23 DUP REM L16 (14 DUPLICATES REMOVED)

=> d ibib abs 1-10

L17 ANSWER 1 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001026433 EMBASE
TITLE: Fetal androgen exposure inhibits fetal rat lung fibroblast lipid uptake and release.
AUTHOR: Rodriguez A.; Viscardi R.M.; Torday J.S.
CORPORATE SOURCE: Dr. A. Rodriguez, Department of Pediatrics, Mercy Medical Center, 301 St. Paul's Place, Baltimore, MD 21202, United States. andres2@home.com
SOURCE: Experimental Lung Research, (2001) 27/1 (13-24).
Refs: 30
ISSN: 0190-2148 CODEN: EXLRDA
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
021 Developmental Biology and Teratology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Fetal lung fibroblasts provide lipid substrate for type II cell surfactant
phospholipid synthesis. This process is developmental and glucocorticoid dependent. Previous studies in our laboratory demonstrating **sex** differences in several aspects of lung maturation suggest that these differences may be due to effects of fetal androgens. Based on these

studies, we hypothesized that fetal lung fibroblast triglyceride metabolism is determined by opposing effects of fetal androgens and glucocorticoids. To model the effects of androgens on fetal lung fibroblast triglyceride metabolism, pregnant rats were treated with dihydrotestosterone (DHT) 1 mg/kg/day from the days 15 to 20 of gestation, and changes in triglyceride content of freshly isolated fetal rat lung fibroblasts (FRLF) and rates of uptake and prostaglandin F(2) (**PGE** (2))-mediated release by cultured FRLF in **response** to glucocorticoids in the presence or absence of DHT in vitro were measured. During lung development, the triglyceride content and rate of uptake of **female**-derived FRLF increased 3.5- and 4.8-fold, respectively, between days 18 and 20 of gestation. From days 19 to 22, male FRLF trigyclyceride content and rate of uptake were lower than the content and uptake by **female** FRLF. Maternal DHT treatment inhibited the normal developmental increase in fibroblast triglyceride content and rate of uptake between days 19 and 22 by both male and **female** FRLF. In the absence of maternal DHT, in vitro dexamethasone stimulated triglyceride uptake 3-fold by day 21 in FRLF. This effect was blocked by maternal pretreatment with DHT. Maternal DHT exposure prevented stimulation of triglyceride release by PGF(2). Although in vitro dexamethasone stimulated triglyceride release by maternal DHT-exposed fibroblasts, it did not enhance the **response** to **PGE** (2). These data suggest that in utero exposure to androgens (1) delay the developmental increase in triglyceride content and (2) oppose the effects of glucocorticoid on cultured FRLF triglyceride uptake and **PGE** (2)-mediated release.

L17 ANSWER 2 OF 23 MEDLINE
ACCESSION NUMBER: 2000027129 MEDLINE
DOCUMENT NUMBER: 20027129 PubMed ID: 10559400
TITLE: Neuregulins signaling via a glial erbB-2-erbB-4 receptor complex contribute to the neuroendocrine control of mammalian sexual development.
AUTHOR: Ma Y J; Hill D F; Creswick K E; Costa M E; Cornea A; Lioubin M N; Plowman G D; Ojeda S R
CORPORATE SOURCE: Division of Neuroscience, Oregon Regional Primate Research Center, Beaverton, Oregon 97006, USA.
CONTRACT NUMBER: HD25123 (NICHD)
P30 HD18185 (NICHD)
RR00163 (NCRR)
SOURCE: JOURNAL OF NEUROSCIENCE, (1999 Nov 15) 19 (22) 9913-27.
Journal code: JDF; 8102140. ISSN: 0270-6474.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199912
ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991202

AB Activation of erbB-1 receptors by glial TGFalpha has been shown to be a component of the developmental program by which the neuroendocrine brain controls mammalian **sexual** development. The participation of other members of the erbB family may be required, however, for full signaling capacity. Here, we show that activation of astrocytic erbB-2/erbB-4 receptors plays a significant role in the process by which the hypothalamus controls the advent of mammalian **sexual** maturation. Hypothalamic astrocytes express both the erbB-2 and erbB-4 genes, but no erbB-3, and **respond** to neuregulins (NRGs) by

releasing prostaglandin E(2) (**PGE**(2)), which acts on neurosecretory neurons to stimulate secretion of luteinizing hormone-releasing hormone (LHRH), the neuropeptide controlling **sexual** development. The actions of TGFalpha and NRGs in glia are synergistic and involve recruitment of erbB-2 as a coreceptor, via erbB-1 and erbB-4, respectively. Hypothalamic expression of both erbB-2 and erbB-4 increases first in a gonad-independent manner before the onset of puberty, and then, at the time of puberty, in a **sex** steroid-dependent manner. Disruption of erbB-2 synthesis in hypothalamic astrocytes by treatment with an antisense oligodeoxynucleotide inhibited the astrocytic **response** to NRGs and, to a lesser extent, that to TGFalpha and blocked the erbB-dependent, glia-mediated, stimulation of LHRH release. Intracerebral administration of the oligodeoxynucleotide to developing animals delayed the initiation of puberty. Thus, activation of the erbB-2-erbB-4 receptor complex appears to be a critical component of the signaling process by which astrocytes facilitate the acquisition of **female** reproductive capacity in mammals.

L17 ANSWER 3 OF 23 MEDLINE
ACCESSION NUMBER: 1999384152 MEDLINE
DOCUMENT NUMBER: 99384152 PubMed ID: 10453054
TITLE: Glial-neuronal interactions in the neuroendocrine control of mammalian puberty: facilitatory effects of gonadal steroids.
AUTHOR: Ojeda S R; Ma Y J
CORPORATE SOURCE: Division of Neuroscience, Oregon Regional Primate Research Center/Oregon Health Sciences University, 505 N.W. 185th Avenue, Beaverton, Oregon 97006, USA.
CONTRACT NUMBER: HD25123 (NICHD)
P30 HD18185 (NICHD)
RR00163 (NCRR)
SOURCE: JOURNAL OF NEUROBIOLOGY, (1999 Sep 15) 40 (4) 528-40.
Ref: 95
Journal code: JAM; 0213640. ISSN: 0022-3034.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 19991014
Last Updated on STN: 19991014
Entered Medline: 19991004

AB It is now clear that astroglial cells actively contribute to both the generation and flow of information within the central nervous system. In the hypothalamus, astrocytes regulate the secretory activity of neuroendocrine neurons. A small subset of these neurons secrete luteinizing hormone-releasing hormone (LHRH), a neuropeptide essential for **sexual** development and adult reproductive function. Astrocytes stimulate LHRH secretion via cell-cell signaling mechanisms involving growth factors recognized by receptors with either serine/threonine or tyrosine kinase activity. Two members of the epidermal growth factor (EGF) family and their respective tyrosine kinase receptors appear to play key roles in this regulatory process. Transforming growth factor-alpha (TGFalpha) and its distant congeners, the neuregulins (NRGs), are produced

in hypothalamic astrocytes. They stimulate LHRH secretion indirectly, via activation of erbB-1/erbB-2 and erbB-4/erbB-2 receptor complexes also located on astrocytes. Activation of these receptors leads to release of prostaglandin E(2) (**PGE**(2)), which then binds to specific receptors on LHRH neurons to elicit LHRH secretion. Gonadal steroids facilitate this glia-to-neuron communication process by acting at three different steps along the signaling pathway. They (a) increase astrocytic gene expression of at least one of the EGF-related ligands (TGFalpha), (b) increase expression of at least two of the receptors (erbB-4 and erbB-2), and (c) enhance the LHRH **response** to **PGE**(2) by up-regulating in LHRH neurons the expression of specific **PGE**(2) receptor isoforms. Focal overexpression of TGFalpha in either the median eminence or preoptic area of the hypothalamus accelerates puberty. Conversely, blockade of either TGFalpha or NRG hypothalamic actions delays the process. Thus, both TGFalpha and NRGs appear to be physiological components of the central neuroendocrine mechanism controlling the initiation of **female** puberty. By facilitating growth factor signaling pathways in the hypothalamus, ovarian steroids accelerate the pace and progression of the pubertal process.

L17 ANSWER 4 OF 23 MEDLINE DUPLICATE 1

ACCESSION NUMBER: 1999264150 MEDLINE
DOCUMENT NUMBER: 99264150 PubMed ID: 10333355
TITLE: Seminal plasma components stimulate interleukin-8 and interleukin-10 release.
AUTHOR: Denison F C; Grant V E; Calder A A; Kelly R W
CORPORATE SOURCE: Department of Obstetrics and Gynaecology, University of Edinburgh, Centre for Reproductive Biology, UK.
SOURCE: MOLECULAR HUMAN REPRODUCTION, (1999 Mar) 5 (3) 220-6.
Journal code: CWO; 9513710. ISSN: 1360-9947.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990806
Last Updated on STN: 19990806
Entered Medline: 19990723

AB Human seminal plasma has potent anti-inflammatory properties which are thought to confer a survival advantage to the spermatozoa within the hostile **female genital** tract. In contrast, a profound pro-inflammatory leukocytosis has been observed post-coitus in animals and humans. Whether components of seminal plasma are involved in initiating this leukocytic reaction is not known. This study investigated the effect of human seminal plasma, a seminal plasma fraction and its principal constituent prostaglandins, prostaglandin E2 (PGE2) and 19-hydroxy **PGE**, on the release of the pro-inflammatory neutrophil chemotactic factor interleukin-8 (IL-8) and the anti-inflammatory cytokines interleukin-10 (IL-10) and secretory leukocyte protease inhibitor (SLPI). The tissues studied were non-pregnant cervical explants, peripheral blood and the monocyte cell line U937. Seminal plasma fraction (SPF) significantly ($P < 0.05$) stimulated release of IL-8 and inhibited release of SLPI from non-pregnant cervical explants. SPF, PGE2 and 19-hydroxy **PGE** significantly ($P< 0.005$) stimulated IL-8 release from peripheral blood and U937 cells. 19-hydroxy **PGE** was significantly ($P< 0.005$) more effective than PGE2 in stimulating IL-8

release. Seminal plasma, SPF and PGE2 significantly ($P < 0.05$) stimulated IL-10 release from U937 cells. 19-hydroxy **PGE** stimulated IL-10 release from U937 cells but this failed to reach significance. Release of IL-10 by cervical explants and SLPI by peripheral blood and U937 cells were below the detection limit of the assays employed. We suggest that the anti- and pro-inflammatory immune **responses** which seminal plasma induces might act in combination initially to promote sperm survival and then to facilitate their removal from the **female genital** tract.

L17 ANSWER 5 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1997:299489 BIOSIS
DOCUMENT NUMBER: PREV199799598692
TITLE: Differential ventral septal vasopressin release is associated with sexual dimorphism in PGE-2 fever.
AUTHOR(S): Chen, X. (1); Landgraf, R.; Pittman, Q. J.
CORPORATE SOURCE: (1) Neuroscience Res. Group, Dep. Physiol. Biophysics, Univ. Calgary, 3330 Hospital Dr. NW, Calgary, AB T2N 4N1 Canada
SOURCE: American Journal of Physiology, (1997) Vol. 272, No. 5 PART 2, pp. R1664-R1669.
ISSN: 0002-9513.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The vasopressinergic innervation of the ventral septal area (VSA) has been shown to be implicated in antipyresis. Because this system is less well developed in **female** rats, we hypothesized that **female** rats would display exaggerated febrile **responses**. We therefore examined the temperature **responses** of conscious and urethan-anesthetized rats of both **sexes** to centrally administered prostaglandin E-2 (**PGE**-2) and correlated these **responses** with the release and action of endogenous arginine vasopressin (AVP) in the VSA. Both conscious (25 ng/5 mu-l **PGE**-2 intracerebroventricularly (icv)) and anesthetized (VSA microdialyzed, 50 ng/5 mu-l **PGE**-2 icv) **female** rats had higher fevers than did males. Infusion of an AVP V-1a receptor antagonist (1 nmol (d(CH-2)-5Tyr(Me))AVP) plus **PGE**-2 gave rise to higher fevers in males but not in **females**. Measurements of AVP in microdialysates of the VSA showed that the release of endogenous AVP was increased in **response** to **PGE**-2 in males only. Baseline AVP release in both **sexes** was similar. The results suggest that there is a **sex**-related difference in **PGE**-2 fever, which may be accounted for by the differential AVP release in the VSA.

L17 ANSWER 6 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1997:449248 BIOSIS
DOCUMENT NUMBER: PREV199799748451
TITLE: Enhancement by sex hormones of the osteoregulatory effects of mechanical loading and prostaglandins in explants of rat ulnae.
AUTHOR(S): Cheng, Ming Zhao; Zaman, Gul; Rawlinson, Simon C. F.; Pitsillides, Andrew A.; Suswillo, Rosemary F. L.; Lanyon, Lance E. (1)
CORPORATE SOURCE: (1) Royal Vet. College, Univ. London, Royal College St., London NW1 0TU UK
SOURCE: Journal of Bone and Mineral Research, (1997) Vol. 12, No.

9, pp. 1424-1430.
ISSN: 0884-0431.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Explants of ulnae from 5-week-old male and **female** rats were cleaned of marrow and soft tissue and, in the presence and absence of 10-8 M 17-beta-estradiol (E2) or 5-alpha-dihydrotestosterone (DHT), mechanically loaded or treated with exogenous prostanoids previously shown to be produced during loading. Over an 18-h period, mechanical loading (peak strain 1300 mu-epsilon, 1 Hz, 8 minutes, maximum strain rate 25,000 mu-epsilon/s), prostaglandin E-2 (**PGE**-2) and prostacyclin (PGI-2) (10-6 M), each separately produced quantitatively similar increases in cell proliferation and matrix production in bones from mates and **females**, as indicated by incorporation of (3H)thymidine into DNA and (3H)proline into collagen. E2 and DHT both increased (3H)thymidine and (3H)proline incorporations, E2 producing greater increases in **females** than in males. Indomethacin abrogated the effects of loading, but had no effects on those of **sex** hormones. Loading, or prostanoids, together with **sex** hormones, produced **responses** generally equal to or greater than the addition of the individual influences acting independently. In **females** there was a synergistic **rèsponse** in (3H)thymidine incorporation between loading and E2, which was quantitatively similar to the interaction between E2 and **PGE**-2 or PGI-2. The interaction between loading and E-2 for (3H)proline incorporation was not mimicked by these prostanoids. In males the synergism in (3H) proline incorporation seen between loading and DHT was mimicked by that between PGI-2 and DHT. We conclude that loading stimulates increased bone cell proliferation and matrix production in situ through a prostanoid-dependent mechanism. This **response** is equal in size in males and **females**. Estrogen and testosterone increase proliferation and matrix production through a mechanism independent of prostanoid production. The interactions between loading and hormones are reproduced in some but not all cases by E-2 and prostaglandins. E-2 with loading and prostaglandins has greater effects in **female** bones, while DHT with loading and prostaglandins has greater effects in males.

L17 ANSWER 7 OF 23 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97302992 MEDLINE
DOCUMENT NUMBER: 97302992 PubMed ID: 9159423
TITLE: A cytokine switch induced by human seminal plasma: an immune modulation with implications for sexually transmitted disease.
AUTHOR: Kelly R W; Carr G G; Critchley H O
CORPORATE SOURCE: Medical Research Council Reproductive Biology Unit, University of Edinburgh Centre for Reproductive Biology, UK.
SOURCE: HUMAN REPRODUCTION, (1997 Apr) 12 (4) 677-81.
Journal code: HRP; 8701199. ISSN: 0268-1161.
PUB. COUNTRY: ENGLAND: United Kingdom
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970805

Last Updated on STN: 19970805
Entered Medline: 19970722

AB The immunosuppressive activity of human seminal plasma may be one factor in the aetiology of **sexually** transmitted disease and could be particularly important for the spread of human immunodeficiency virus (HIV). The advent of virus that can preferentially infect Langerhans cells of the **genital** mucosa underscores the relevance of seminal plasma effects. Virally infected cells are eradicated by the killing activity of T cells and natural killer (NK) cells and this cytotoxicity is stimulated by IL-12 (previously known as natural killer cell stimulatory factor) and partly inhibited by IL-10 (previously known as cytokine synthesis inhibitory factor). We have examined the effects of human seminal plasma on the production of these key cytokines. Cytokine production was measured in rapidly diluted, fresh, lipopolysaccharide (LPS)-stimulated, whole blood since this provided leukocytes with minimal exposure to prostaglandin. Prostaglandin concentrations and cytokine release were measured by ELISA. Addition of human seminal plasma diluted up to 100,000 times (0.001%) to blood cell cultures led to a marked increase in the IL-10/IL-12 ratio ($P < 0.02$). A dose-dependent increase in the ratio was observed in five separate experiments, from a control value of 1 (no seminal plasma) to a mean value of 80 (1% seminal plasma). This cytokine switch was also seen when seminal plasma was substituted by pure prostaglandin E (**PGE**) and 19-OH **PGE** (the main prostaglandin constituent of human seminal plasma). Lipid-extracted seminal plasma was considerably less active at high dilutions than whole seminal plasma at the same dilution. However, its activity could be restored by the addition of synthetic **PGE** and 19-hydroxy **PGE**. A stimulation of IL-10 and a decrease in IL-12 in host-defence cells of the lower **female** reproductive tract will seriously affect the ability of cytotoxic T cells and NK cells to recognise and destroy virally infected cells. In addition, the stimulation of IL-10 will inhibit the release of the anti-HIV activity from CD8+ve cells. The cytokine switch reported here, activated by semen deposition, would exercise a key inhibitory control over vital immune defences in the lower **genital** tract, with ablation of cell-mediated **responses** and immunosurveillance.

L17 ANSWER 8 OF 23 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1998041214 MEDLINE
DOCUMENT NUMBER: 98041214 PubMed ID: 9373880
TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women.
AUTHOR: Farker K; Schweer H; Vollandt R; Nassr N; Nagel U; Seyberth H W; Hoffmann A; Oettel M
CORPORATE SOURCE: Institute of Clinical Pharmacology, Friedrich Schiller University, Jena, Germany.
SOURCE: PROSTAGLANDINS, (1997 Sep) 54 (3) 655-64.
Journal code: Q76; 0320271. ISSN: 0090-6980.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19980109

Entered Medline: 19971218

AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E2 (PGE2), 6-keto-PGF1 alpha, thromboxane B2 (TxB2) and their metabolites **PGE**-M (11 alpha-hydroxy-9, 15-dioxo-2,3,4,5,20-pentanor-19-carboxyprostanoic acid), 2,3-dinor-6-keto-PGF1 alpha, 2,3-dinor-TxB2 and 11-dehydro-TxB2 were determined by gas chromatography-triple stage quadrupole mass spectrometry (GC/MS/MS) in 41 young non-pregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women**. Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, PGE2 and TxB2 were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels of estradiol and progesterone were different in young and postmenopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins. Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

L17 ANSWER 9 OF 23 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 97115452 MEDLINE
DOCUMENT NUMBER: 97115452 PubMed ID: 8956853
TITLE: Self-reported sexual function and sexual arousability in women with epilepsy.
AUTHOR: Morrell M J; Guldner G T
CORPORATE SOURCE: Department of Neurology and Neurological Sciences, Stanford
Comprehensive Epilepsy Center, Stanford University School of Medicine, California, USA.
SOURCE: EPILEPSIA, (1996 Dec) 37 (12) 1204-10.
Journal code: EIX; 2983306R. ISSN: 0013-9580.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19990129
Entered Medline: 19970116

AB PURPOSE: **Women** with epilepsy are at risk for **sexual** dysfunction but the frequency and types of dysfunction have not been well characterized. METHODS: Self-reported **sexual** function was evaluated in 116 **women** aged 18-65 years with epilepsy and no concomitant medical or psychiatric illness, including 99 with localization-related epilepsy (LRE) and 17 with primary generalized epilepsy (**PGE**). Variables evaluated included seizure frequency, age of seizure onset, and antiepileptic drug (AED) exposure. Standardized inventories assessed **sexual** functioning, **sexual** arousability and anxiety, **sexual** behavior, and depression. RESULTS: Although **sexual** experience was not reduced, **women** with **PGE** and LRE reported significantly less

sexual arousability and **women** with LRE reported significantly more **sexual** anxiety. **Women** with LRE experienced significantly more dyspareunia, vaginismus, **arousal** insufficiency, and **sexual** dissatisfaction, whereas **women** with **PGE** experienced anorgasmia and **sexual** dissatisfaction. **Sexual** symptoms were not associated with seizure frequency, AED exposure, **sexual** experience, depression, or prepubertal seizure onset. CONCLUSIONS: In contrast to subjects of previous research, the **women** in our study did not have a disorder of **sexual desire**, but more than one third experienced disorders of **sexual arousal**, implying a physiological deficit. Although the etiology for these **arousal** phase dysfunctions has not been defined, such conditions are treatable and warrant referral to a gynecologist versed in the treatment of **sexual** disorders.

L17 ANSWER 10 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1996:360368 BIOSIS
DOCUMENT NUMBER: PREV199699082724
TITLE: Progesterone and dexamethasone stimulate proliferation and differentiation of osteoprogenitors and progenitors for adipocytes and macrophages in cell populations derived from adult rat vertebrae.
AUTHOR(S): Ishida, Yoichiro (1); Tertinegg, Inka; Heersche, Johan N. M.
CORPORATE SOURCE: (1) Fac. Dentistry, Univ. Toronto, Room 400, 124 Edward Street, Toronto, ON M5G 1G6 Canada
SOURCE: Journal of Bone and Mineral Research, (1996) Vol. 11, No. 7, pp. 921-930.
ISSN: 0884-0431.
DOCUMENT TYPE: Article
LANGUAGE: English
AB We investigated the effects of the **sex** hormone progesterone (Prog) and the synthetic glucocorticoid dexamethasone (Dex) on proliferation and differentiation of progenitor cells of osteogenic, adipocytic, and hemopoietic lineages in cell populations derived from explants of adult **female** rat lumbar vertebrae. The cell populations were obtained by culturing bone explants in plasma clots immersed in alpha-minimum essential medium plus 10% fetal calf serum (standard medium) and then subculturing the outgrowth cells in standard medium plus 50 mu-g/ml of ascorbic acid, 5 mM beta-glycerophosphate, and with or without Prog or Dex. On day 6 of culture, these populations were analyzed for cAMP **responses** to parathyroid hormone (PTH), prostaglandin E-2 (**PGE**-2), and isoproterenol (IPT). Increases in intracellular cAMP were seen in **response** to PTH, **PGE**-2, and IPT, and culturing in medium containing Prog increased these **responses**. At various time periods between days 4-27 of culture, the cultures were evaluated for the presence of bone nodules, alkaline phosphatase (AP)-positive colonies, adipocytes, monocytes, and macrophages. Prog and Dex increased the number of bone nodules and AP-positive colonies. The effect of Prog on bone nodule formation was smaller than that of Dex. In addition, the effect of Dex on bone nodule formation was evident after 10 days of culture, while the Prog-induced effects became significant at days 16-20 of culture. Both hormones also increased the number of Sudan IV-positive colonies (adipocytes), certain types of alpha-naphthyl butyrate esterase (alpha-NBE)-positive colonies (monocytes, macrophages, and T-lymphocytes), and ED-2-positive colonies (macrophages). Prog-treated cultures contained more colonies of small

spindle-shaped alpha-NBE-positive cells and fewer colonies of small round a-NBE-positive cells when compared with Dex-treated cultures. These data indicate that cell populations derived from adult rat lumbar vertebrae contain, among. others, osteoprogenitors and progenitors for adipocytes and macrophages that are stimulated to proliferate and differentiate by Prog and Dex. The data also suggest that the effects of Prog and Dex differ qualitatively and quantitatively.

=> s l17 range=, 1999
L18 21 L17

=> s l17 range=, 1998
L19 19 L17

=> d ti so tot

L19 ANSWER 1 OF 19 MEDLINE
TI Measurements of urinary prostaglandins in young ovulatory women during the
menstrual cycle and in postmenopausal women.
SO PROSTAGLANDINS, (1997 Sep) 54 (3) 655-64.
Journal code: Q76; 0320271. ISSN: 0090-6980.

L19 ANSWER 2 OF 19 MEDLINE
TI A cytokine switch induced by human seminal plasma: an immune modulation with implications for sexually transmitted disease.
SO HUMAN REPRODUCTION, (1997 Apr) 12 (4) 677-81.
Journal code: HRP; 8701199. ISSN: 0268-1161.

L19 ANSWER 3 OF 19 MEDLINE
TI Self-reported sexual function and sexual arousability in women with epilepsy.
SO EPILEPSIA, (1996 Dec) 37 (12) 1204-10.
Journal code: EIX; 2983306R. ISSN: 0013-9580.

L19 ANSWER 4 OF 19 MEDLINE
TI [Prostaglandins and reproduction. I. Physiological aspects].
Prostaglandines et reproduction. I. Aspects physiologiques.
SO JOURNAL DE GYNECOLOGIE, OBSTETRIQUE ET BIOLOGIE DE LA REPRODUCTION, (1991)
20 (1) 7-17. Ref: 69
Journal code: IAZ; 0322206. ISSN: 0368-2315.

L19 ANSWER 5 OF 19 MEDLINE
TI Effects of female sex hormones and pregnancy on gallbladder prostaglandin synthesis.
SO ARCHIVES OF SURGERY, (1988 Jun) 123 (6) 705-8.
Journal code: 8IA; 9716528. ISSN: 0004-0010.

L19 ANSWER 6 OF 19 MEDLINE
TI Prostaglandin-mediated inhibition of lymphokine secretion in normal individuals and patients with progressive systemic sclerosis (scleroderma,
PSS).
SO AGENTS AND ACTIONS, (1982 Oct) 12 (4) 471-7.
Journal code: 2XZ; 0213341. ISSN: 0065-4299.

L19 ANSWER 7 OF 19 MEDLINE
TI How important are prostaglandins in the urology of man?.

SO UROLOGIA INTERNATIONALIS, (1982) 37 (3) 160-71.
Journal code: WRI; 0417373. ISSN: 0042-1138.

L19 ANSWER 8 OF 19 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
TI Effect of adrenoceptor blockers and maoi on postcoital utero-oviductal contractility, sperm transport, and sperm attachment to eggs in rabbits.
SO Archives of Andrology, (1981) 6/4 (307-316).
CODEN: ARANDR

L19 ANSWER 9 OF 19 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
TI Effects of cannabinoids and female exposure on the pituitary testicular axis in mice: Possible involvement of prostaglandins.
SO Biology of Reproduction, (1981) 24/2 (315-322).
CODEN: BIREBV

L19 ANSWER 10 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Enhancement by sex hormones of the osteoregulatory effects of mechanical loading and prostaglandins in explants of rat ulnae.
SO Journal of Bone and Mineral Research, (1997) Vol. 12, No. 9, pp. 1424-1430.
ISSN: 0884-0431.

L19 ANSWER 11 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Differential ventral septal vasopressin release is associated with sexual dimorphism in PGE-2 fever.
SO American Journal of Physiology, (1997) Vol. 272, No. 5 PART 2, pp. R1664-R1669.
ISSN: 0002-9513.

L19 ANSWER 12 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Progesterone and dexamethasone stimulate proliferation and differentiation
of osteoprogenitors and progenitors for adipocytes and macrophages in cell
populations derived from adult rat vertebrae.
SO Journal of Bone and Mineral Research, (1996) Vol. 11, No. 7, pp. 921-930.
ISSN: 0884-0431.

L19 ANSWER 13 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Influence of the estrous cycle on the norepinephrine-induced contraction of rat aorta: Relationship to vascular prostanoids biosynthesis.
SO Biological Research, (1994) Vol. 27, No. 3-4, pp. 209-215.
ISSN: 0716-9760.

L19 ANSWER 14 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Mesenteric vascular responses to vasopressin during development of DOCA-salt hypertension in male and female rats.
SO American Journal of Physiology, (1995) Vol. 268, No. 1 PART 2, pp. R40-R49.
ISSN: 0002-9513.

L19 ANSWER 15 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Gender-related response to a tert-butyl hydroperoxide-induced oxidation in
human neonatal tissue.
SO Free Radical Biology & Medicine, (1994) Vol. 16, No. 3, pp. 307-313.
ISSN: 0891-5849.

L19 ANSWER 16 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS

TI Sexually dimorphic effects of alcohol exposure in utero on neuroendocrine and immune functions in chronic alcohol-exposed adult rats.
SO Molecular and Cellular Neuroscience, (1993) Vol. 4, No. 4, pp. 343-353. ISSN: 1044-7431.

L19 ANSWER 17 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI ROLE OF PROSTAGLANDINS IN FISH REPRODUCTION.
SO CAN J FISH AQUAT SCI, (1982) 39 (1), 92-98.
CODEN: CJFSDX. ISSN: 0706-652X.

L19 ANSWER 18 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI INFLUENCE OF SEX AND AGE ON FEBRILE RESPONSES TO PERIPHERAL AND CENTRAL ADMINISTRATION OF PYROGENS IN THE RABBIT.
SO J PHYSIOL (LOND), (1979) 295 (0), 263-272.
CODEN: JPHYA7. ISSN: 0022-3751.

L19 ANSWER 19 OF 19 USPATFULL
TI Synergistic composition comprising PGF.sub.2.sub..alpha. and PGE.sub.2

=> fil stng

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	43.70	94.52

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'STNGUIDE' ENTERED AT 12:16:42 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May 11, 2001 (20010511/UP).

=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.00	94.52

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, ...' ENTERED AT 12:20:12 ON 14 MAY 2001

59 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view search error messages that display as 0* with SET DETAIL OFF.

=> s l1 or misoprostol or (prostagladin E) or PGE
1241* FILE ADISALERTS

```
       39   FILE ADISINSIGHT
       81   FILE AGRICOLA
       24   FILE ANABSTR
      104*  FILE AQUASCI
      172   FILE BIOBUSINESS
        2*  FILE BIOCOMMERCE
     9147   FILE BIOSIS
       15   FILE BIOTECHABS
       15   FILE BIOTECHDS
     4490   FILE BIOTECHNO
      439*  FILE CABA
      808   FILE CANCERLIT
     5060*  FILE CAPLUS
       35*  FILE CEABA-VTB
        1   FILE CEN
       76   FILE CIN
      361*  FILE CONFSCI
        0*  FILE CROPB
        2*  FILE CROPU
      120*  FILE DDFB
     2488*  FILE DDFU
22 FILES SEARCHED...
       61*  FILE DGENE
      120*  FILE DRUGB
      121   FILE DRUGLAUNCH
      125   FILE DRUGMONOG2
       19   FILE DRUGNL
     2874*  FILE DRUGU
       14   FILE DRUGUPDATES
      171*  FILE EMBAL
     5933   FILE EMBASE
     3570*  FILE ESBIOBASE
        0*  FILE FOMAD
        0*  FILE FOREGE
       11*  FILE FROSTI
       14   FILE FSTA
       25*  FILE GENBANK
       26*  FILE HEALSAFE
      259*  FILE IFIPAT
      282   FILE JICST-EPLUS
        3*  FILE KOSMET
     3307*  FILE LIFESCI
        1*  FILE MEDICONF
43 FILES SEARCHED...
     4475   FILE MEDLINE
       45   FILE NIOSHTIC
       84*  FILE NTIS
       23*  FILE OCEAN
     5188*  FILE PASCAL
       30   FILE PHAR
        0*  FILE PHIC
      476*  FILE PHIN
     1371   FILE PROMT
     8868*  FILE SCISEARCH
        6   FILE SYNTHLINE
     1693   FILE TOXLINE
     2279   FILE TOXLIT
     3435*  FILE USPATFULL
      375   FILE WPIDS
58 FILES SEARCHED...
```

```
      375   FILE WPINDEX

  55 FILES HAVE ONE OR MORE ANSWERS,   59 FILES SEARCHED IN STNINDEX

L20  QUE L1 OR MISOPROSTOL OR (PROSTAGLADIN E) OR PGE

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia) )
        283   FILE ADISALERTS
         69   FILE ADISINSIGHT
        832   FILE AGRICOLA
          2   FILE ANABSTR
        734   FILE AQUASCI
        303   FILE BIOBUSINESS
          2   FILE BIOCOMMERCE
      15038   FILE BIOSIS
         12   FILE BIOTECHABS
   9 FILES SEARCHED...
         12   FILE BIOTECHDS
       1825   FILE BIOTECHNO
       4136   FILE CABA
       2254   FILE CANCERLIT
  13 FILES SEARCHED...
       1933   FILE CAPLUS
          2   FILE CEABA-VTB
         11   FILE CEN
          9   FILE CIN
         23   FILE CONFSCI
        117   FILE CROPB
        374   FILE CROPU
         96   FILE DDFB
        421   FILE DDFU
  22 FILES SEARCHED...
        100   FILE DGENE
         96   FILE DRUGB
          8   FILE DRUGNL
       1532   FILE DRUGU
         14   FILE DRUGUPDATES
        152   FILE EMBAL
      13934   FILE EMBASE
  31 FILES SEARCHED...
       3509   FILE ESBIOBASE
          2   FILE FOMAD
         21   FILE FROSTI
         61   FILE FSTA
          1   FILE GENBANK
  37 FILES SEARCHED...
        231   FILE HEALSAFE
         57   FILE IFIPAT
        748   FILE JICST-EPLUS
         15   FILE KOSMET
       4182   FILE LIFESCI
          2   FILE MEDICONF
      13531   FILE MEDLINE
  44 FILES SEARCHED...
        561   FILE NIOSHTIC
        347   FILE NTIS
        185   FILE OCEAN
       4100   FILE PASCAL
```

```
          9   FILE PHAR
  49 FILES SEARCHED...
          1   FILE PHIC
         64   FILE PHIN
       1233   FILE PROMT
       7404   FILE SCISEARCH
       3855   FILE TOXLINE
  55 FILES SEARCHED...
       3780   FILE TOXLIT
        943   FILE USPATFULL
         91   FILE WPIDS
         91   FILE WPINDEX

  55 FILES HAVE ONE OR MORE ANSWERS,    59 FILES SEARCHED IN STNINDEX

L21  QUE (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR GENITAL)
(S)
         (HYPOACTIV? OR DESIR? OR SATISFACT? OR ORGASM OR AROUSAL? OR
SENSATION
         ? OR RESPON? OR ANHEDONIA) )

=> s l20 and l21
          1*  FILE ADISALERTS
          1   FILE ADISINSIGHT
          0*  FILE AQUASCI
          0*  FILE BIOCOMMERCE
         19   FILE BIOSIS
  10 FILES SEARCHED...
          8   FILE BIOTECHNO
          2*  FILE CABA
          4   FILE CANCERLIT
          7*  FILE CAPLUS
  14 FILES SEARCHED...
          0*  FILE CEABA-VTB
          0*  FILE CONFSCI
          0*  FILE CROPB
          0*  FILE CROPU
          0*  FILE DDFB
          1*  FILE DDFU
          0*  FILE DGENE
          0*  FILE DRUGB
          3*  FILE DRUGU
          1   FILE DRUGUPDATES
  29 FILES SEARCHED...
          0*  FILE EMBAL
         11   FILE EMBASE
          8*  FILE ESBIOBASE
          0*  FILE FOMAD
          0*  FILE FOREGE
          0*  FILE FROSTI
          0*  FILE GENBANK
  37 FILES SEARCHED...
          0*  FILE HEALSAFE
          0*  FILE IFIPAT
          0*  FILE KOSMET
          6*  FILE LIFESCI
          0*  FILE MEDICONF
         10   FILE MEDLINE
  45 FILES SEARCHED...
          0*  FILE NTIS
```

```
          0*  FILE OCEAN
         10*  FILE PASCAL
          0*  FILE PHIC
          0*  FILE PHIN
          5   FILE PROMT
  52 FILES SEARCHED...
         18*  FILE SCISEARCH
          2   FILE TOXLINE
          8   FILE TOXLIT
         15*  FILE USPATFULL
  57 FILES SEARCHED...
          1   FILE WPIDS
          1   FILE WPINDEX

  22 FILES HAVE ONE OR MORE ANSWERS,    59 FILES SEARCHED IN STNINDEX

L22  QUE L20 AND L21

=> d rank
F1           19   BIOSIS
F2           18*  SCISEARCH
F3           15*  USPATFULL
F4           11   EMBASE
F5           10   MEDLINE
F6           10*  PASCAL
F7            8   BIOTECHNO
F8            8   TOXLIT
F9            8*  ESBIOBASE
F10           7*  CAPLUS
F11           6*  LIFESCI
F12           5   PROMT
F13           4   CANCERLIT
F14           3*  DRUGU
F15           2   TOXLINE
F16           2*  CABA
F17           1   ADISINSIGHT
F18           1   DRUGUPDATES
F19           1   WPIDS
F20           1   WPINDEX
F21           1*  ADISALERTS
F22           1*  DDFU


=> file f2, f6-f9, f11-f22
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                 5.40      99.92

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
                                                   ENTRY    SESSION
CA SUBSCRIBER PRICE                                 0.00      -0.59

FILE 'SCISEARCH' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Institute for Scientific Information (ISI) (R)

FILE 'PASCAL' ENTERED AT 12:27:36 ON 14 MAY 2001
Any reproduction or dissemination in part or in full,
by means of any process and on any support whatsoever
is prohibited without the prior written agreement of INIST-CNRS.
COPYRIGHT (C) 2001 INIST-CNRS. All rights reserved.
```

FILE 'BIOTECHNO' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'TOXLIT' ENTERED AT 12:27:36 ON 14 MAY 2001

FILE 'ESBIOBASE' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'LIFESCI' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Cambridge Scientific Abstracts (CSA)

FILE 'PROMT' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Gale Group. All rights reserved.

FILE 'CANCERLIT' ENTERED AT 12:27:36 ON 14 MAY 2001

FILE 'DRUGU' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

FILE 'TOXLINE' ENTERED AT 12:27:36 ON 14 MAY 2001

FILE 'CABA' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 CAB INTERNATIONAL (CABI)

FILE 'ADISINSIGHT' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

FILE 'DRUGUPDATES' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 IMSWORLD Publications Ltd

FILE 'WPIDS' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

FILE 'WPINDEX' ACCESS NOT AUTHORIZED

FILE 'ADISALERTS' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

FILE 'DDFU' ACCESS NOT AUTHORIZED

=> s f22
L23 605 F22

=> s l22
'CN' IS NOT A VALID FIELD CODE
'CN' IS NOT A VALID FIELD CODE
3 FILES SEARCHED...
'CN' IS NOT A VALID FIELD CODE
'CN' IS NOT A VALID FIELD CODE
7 FILES SEARCHED...
10 FILES SEARCHED...
14 FILES SEARCHED...
L24 79 L22

=> s l20 (s) l21
'CN' IS NOT A VALID FIELD CODE
'CN' IS NOT A VALID FIELD CODE
3 FILES SEARCHED...

'CN' IS NOT A VALID FIELD CODE
'CN' IS NOT A VALID FIELD CODE
7 FILES SEARCHED...
9 FILES SEARCHED...
12 FILES SEARCHED...
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L65 (S) L66'
L25 69 L20 (S) L21

=> s cellulose
L26 222051 CELLULOSE

=> s l26 (s) l25
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L129 (S) L113'
L27 0 L26 (S) L25

=> s l26 and l25
L28 0 L26 AND L25

=> d l25 1-5 ibib abs kwic

L25 ANSWER 1 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 2001:59328 SCISEARCH
THE GENUINE ARTICLE: 390QT
TITLE: Fetal androgen exposure inhibits fetal rat lung fibroblast
lipid uptake and release
AUTHOR: Rodriguez A (Reprint); Viscardi R M; Torday J S
CORPORATE SOURCE: Mercy Med Ctr, Dept Pediat, 301 St Pauls Pl, Baltimore, MD
21202 USA (Reprint); Univ Maryland, Sch Med, Dept Pediat, Div Neonatol, Baltimore, MD 21201 USA
COUNTRY OF AUTHOR: USA
SOURCE: EXPERIMENTAL LUNG RESEARCH, (JAN-FEB 2001) Vol. 27, No. 1,
pp. 13-24.
Publisher: HEMISPHERE PUBL CORP, 1900 FROST ROAD, SUITE 101, BRISTOL, PA 19007-1598 USA.
ISSN: 0190-2148.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 30
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Fetal lung fibroblasts provide lipid substrate for the II cell surfactant phospholipid synthesis. This process is developmental and glucocorticoid dependent. Previous studies in our laboratory demonstrating **sex** differences in several aspects of lung maturation suggest that these differences may be due to effects of fetal androgens. Based on these studies, we hypothesi;ed that fetal lung fibroblast triglyceride metabolism is determined by opposing effects of fetal androgens and glucocorticoids. To model the effects of androgens on fetal lung fibroblast triglyceride metabolism, pregnant rats were treated with dihyrdrotestosterone (DHT) 1 mg/kg/day from the days 15 to 20 of gestation and changes in triglyceride content of freshly isolated fetal rat lung fibroblasts (FRLF) and rates of uptake and prostaglandin E-2 (**PGE** (2))-mediated release by cultured FRLF in **response** to

glucocorticoids in the presence or absence of DHT In vitro were measured. During lung development, the triglyceride content and rate of uptake of **female**-derived FRLF increased 3.5- and 4.8-fold, respectively, between days 18 and 20 of gestation. From days 19 to 22, male FRLF trigyclyceride content and rate of uptake were lower than the content and uptake by **female** FRLF. Maternal DHT treatment inhibited the normal developmental increase in fibroblast triglyceride content and rate of uptake between days 19 and 22 by both male and **female** FRLF. In. the absence of maternal DHT, in vitro dexamethasone stimulated triglyceride uptake 3-fold by day 21 in FRLF. This effect was blocked by maternal preteatment with DHT Maternal DHT exposure prevented stimulation of triglyceride release by **PGE**(2). Although in vitro dexamethasone stimulated triglyceride release by maternal DHT-exposed fibroblasts, it did not enhance the **response** to **PGE** (2). These data suggest that in utero exposure to androgens (1) delay the developmental increase in triglyceride content and (2) oppose the effects of glucocorticoid on cultured FRLF triglyceride uptake and **PGE** (2)-mediated release.

AB . . . for the II cell surfactant phospholipid synthesis. This process is developmental and glucocorticoid dependent. Previous studies in our laboratory demonstrating **sex** differences in several aspects of lung maturation suggest that these differences may be due to effects of fetal androgens. Based. . . and changes in triglyceride content of freshly isolated fetal rat lung fibroblasts (FRLF) and rates of uptake and prostaglandin E-2 (**PGE**(2))-mediated release by cultured FRLF in **response** to glucocorticoids in the presence or absence of DHT In vitro were measured. During lung development, the triglyceride content and rate of uptake of **female**-derived FRLF increased 3.5- and 4.8-fold, respectively, between days 18 and 20 of gestation. From days 19 to 22, male FRLF trigyclyceride content and rate of uptake were lower than the content and uptake by **female** FRLF. Maternal DHT treatment inhibited the normal developmental increase in fibroblast triglyceride content and rate of uptake between days 19 and 22 by both male and **female** FRLF. In. the absence of maternal DHT, in vitro dexamethasone stimulated triglyceride uptake 3-fold by day 21 in FRLF. This effect was blocked by maternal preteatment with DHT Maternal DHT exposure prevented stimulation of triglyceride release by **PGE** (2). Although in vitro dexamethasone stimulated triglyceride release by maternal DHT-exposed fibroblasts, it did not enhance the **response** to **PGE**(2). These data suggest that in utero exposure to androgens (1) delay the developmental increase in triglyceride content and (2) oppose the effects of glucocorticoid on cultured FRLF triglyceride uptake and **PGE**(2)-mediated release.

L25 ANSWER 2 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:941986 SCISEARCH
THE GENUINE ARTICLE: 260YL
TITLE: The immunobiology of sexual behavior: Gender differences in the suppression of sexual activity during illness
AUTHOR: Avitsur R; Yirmiya R (Reprint)
CORPORATE SOURCE: HEBREW UNIV JERUSALEM, DEPT PSYCHOL, IL-91905 JERUSALEM, ISRAEL (Reprint); HEBREW UNIV JERUSALEM, DEPT PSYCHOL, IL-91905 JERUSALEM, ISRAEL
COUNTRY OF AUTHOR: ISRAEL

SOURCE: PHARMACOLOGY BIOCHEMISTRY AND BEHAVIOR, (DEC 1999) Vol. 64, No. 4, pp. 787-796.
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND.
ISSN: 0091-3057.
DOCUMENT TYPE: General Review; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 117
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Following infection or injury, sick individuals experience profound psychological and behavioral changes, such as anorexia, depressed activity, and reduced self-care behavior. In the present review, we present evidence for a gender-difference in the behavioral **response** to sickness. Specifically, following immune activation, **sexual** activity is suppressed in **female**, but not in male rats. This gender difference is specific to **sexually** related **responses**, because other behaviors, such as locomotion, are equally affected by immune challenges in males and estrous **females**. The suppression of **female sexual** behavior, induced by either endotoxin (lipopolysaccharide), or the cytokine interleukin-1 (IL-1), are mediated by central mechanisms that are independent of alterations in ovarian hormone secretion. Furthermore, synergistic effects of the cytokines IL-1 and tumor necrosis factor alpha (TNF alpha) are involved in modulating **sexual** behavior in sick **females**, and prostaglandins synthesis is required for the effects of IL-1 on **female sexual** behavior. The gender difference in the behavioral **response** to immune activation may be related to the findings that at the same doses and timing in which IL-1 suppressed **sexual** activity in **female** but not in male rats, **females** produced more prostaglandin E-2 (**PGE**(2)) in the brain, and less corticosterone than males. Finally, we are suggesting that the suppressive effect of cytokines on **female** reproductive behavior may serve as a mechanism to reduce conception during infection, which exposes the mother and the fetus to dangers such as spontaneous abortions, preterm labor and maternal mortality. (C) 1999 Elsevier Science Inc.

AB . . . anorexia, depressed activity, and reduced self-care behavior. In the present review, we present evidence for a gender-difference in the behavioral **response** to sickness. Specifically, following immune activation, **sexual** activity is suppressed in **female**, but not in male rats. This gender difference is specific to **sexually** related **responses**, because other behaviors, such as locomotion, are equally affected by immune challenges in males and estrous **females**. The suppression of **female sexual** behavior, induced by either endotoxin (lipopolysaccharide), or the cytokine interleukin-1 (IL-1), are mediated by central mechanisms that are independent of. . . hormone secretion. Furthermore, synergistic effects of the cytokines IL-1 and tumor necrosis factor alpha (TNF alpha) are involved in modulating **sexual** behavior in sick **females**, and prostaglandins synthesis is required for the effects of IL-1 on **female sexual** behavior. The gender difference in the behavioral **response** to immune activation may be related to the findings that at the same doses and timing in which IL-1 suppressed **sexual** activity in **female** but not in male

rats, **females** produced more prostaglandin E-2 (**PGE**(2)) in the brain, and less corticosterone than males. Finally, we are suggesting that the suppressive effect of cytokines on **female** reproductive behavior may serve as a mechanism to reduce conception during infection, which exposes the mother and the fetus to. . .

L25 ANSWER 3 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:879260 SCISEARCH
THE GENUINE ARTICLE: 254JG
TITLE: Neuregulins signaling via a glial erbB-2-erbB-4 receptor complex contribute to the neuroendocrine control of mammalian sexual development
AUTHOR: Ma Y J; Hill D F; Creswick K E; Costa M E; Cornea A; Lioubin M N; Plowman G D; Ojeda S R (Reprint)
CORPORATE SOURCE: OREGON REG PRIMATE RES CTR, DIV NEUROSCI, 505 NW 185TH AVE, BEAVERTON, OR 97006 (Reprint); OREGON REG PRIMATE RES CTR, DIV NEUROSCI, BEAVERTON, OR 97006; SUGEN INC, S SAN FRANCISCO, CA 94080
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF NEUROSCIENCE, (15 NOV 1999) Vol. 19, No. 22, pp. 9913-9927.
Publisher: SOC NEUROSCIENCE, 11 DUPONT CIRCLE, NW, STE 500, WASHINGTON, DC 20036.
ISSN: 0270-6474.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 77

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Activation of erbB-1 receptors by glial TGF alpha has been shown to be a component of the developmental program by which the neuroendocrine brain controls mammalian **sexual** development. The participation of other members of the erbB family may be required, however, for full signaling capacity. Here, we show that activation of astrocytic erbB-2/erbB-4 receptors plays a significant role in the process by which the hypothalamus controls the advent of mammalian **sexual** maturation. Hypothalamic astrocytes express both the erbB-2 and erbB-4 genes, but no erbB-3, and **respond** to neuregulins (NRGs) by releasing prostaglandin E-2 (**PGE**(2)), which acts on neurosecretory neurons to stimulate secretion of luteinizing hormone-releasing hormone (LHRH), the neuropeptide controlling **sexual** development. The actions of TGF alpha and NRGs in glia are synergistic and involve recruitment of erbB-2 as a coreceptor, via erbB-1 and erbB-4, respectively. Hypothalamic expression of both erbB-2 and erbB-4 increases first in a gonad-independent manner before the onset of puberty, and then, at the time of puberty, in a **sex** steroid-dependent manner. Disruption of erbB-2 synthesis in hypothalamic astrocytes by treatment with an antisense oligodeoxynucleotide inhibited the astrocytic **response** to NRGs and, to a lesser extent, that to TGF alpha and blocked the erbB-dependent, glia-mediated, stimulation of LHRH release. Intracerebral administration of the oligodeoxynucleotide to developing animals delayed the initiation of puberty. Thus, activation of the erbB-2-erbB-4 receptor complex appears to be a critical component of the signaling process by which astrocytes facilitate the acquisition of **female** reproductive capacity in mammals.

AB . . . TGF alpha has been shown to be a component of the developmental program by which the neuroendocrine brain controls mammalian

sexual development. The participation of other members of the erbB family may be required, however, for full signaling capacity. Here, we.

. of astrocytic erbB-2/erbB-4 receptors plays a significant role in the process by which the hypothalamus controls the advent of mammalian **sexual** maturation. Hypothalamic astrocytes express both the erbB-2 and erbB-4 genes, but no erbB-3, and **respond** to neuregulins (NRGs) by releasing prostaglandin E-2 (**PGE**(2)), which acts on neurosecretory neurons to stimulate secretion of luteinizing hormone-releasing hormone (LHRH), the neuropeptide controlling **sexual** development. The actions of TGF alpha and NRGs in glia are synergistic and involve recruitment of erbB-2 as a coreceptor,. . . increases first in a gonad-independent manner before the onset of puberty, and then, at the time of puberty, in a **sex** steroid-dependent manner. Disruption of erbB-2 synthesis in hypothalamic astrocytes by treatment with an antisense oligodeoxynucleotide inhibited the astrocytic **response** to NRGs and, to a lesser extent, that to TGF alpha and blocked the erbB-dependent, glia-mediated, stimulation of LHRH release.. . . erbB-2-erbB-4 receptor complex appears to be a critical component of the signaling process by which astrocytes facilitate the acquisition of **female** reproductive capacity in mammals.

L25 ANSWER 4 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:682829 SCISEARCH
THE GENUINE ARTICLE: 231YE
TITLE: Identification of specific EP receptors responsible for the hemodynamic effects of PGE(2)
AUTHOR: Audoly L P; Tilley S L; Goulet J; Key M; Nguyen M; Stock J L; McNeish J D; Koller B H; Coffman T M (Reprint)
CORPORATE SOURCE: DUKE UNIV, MED CTR, DIV NEPHROL, DEPT MED, BOX 3014, DURHAM, NC 27710 (Reprint); DUKE UNIV, MED CTR, DIV NEPHROL, DEPT MED, DURHAM, NC 27710; DURHAM VET AFFAIRS MED CTR, DURHAM, NC 27710; UNIV N CAROLINA, DEPT MED, CHAPEL HILL, NC 27599; PFIZER INC, CTR EXPT THERAPEUT, GROTON, CT 06340
COUNTRY OF AUTHOR: USA
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-HEART AND CIRCULATORY PHYSIOLOGY, (SEP 1999) Vol. 46, No. 3, pp. H924-H930. Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814. ISSN: 0363-6135.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 39

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB To identify the E-prostanoid (EP) receptors that mediate the hemodynamic actions of **PGE**(2), we studied acute vascular **responses** to infusions of **PGE**(2) using lines of mice in which each of four EP receptors (EP1 through EP4) have been disrupted by gene targeting. In mixed groups of males and **females**, vasodepressor **responses** after infusions of **PGE**(2) were significantly diminished in the EP2 (-/-) and EP4 (-/-) lines but not in the EP1 -/- or EP3 -/- lines. Because the actions of other hormonal systems that regulate blood pressure differ between **sexes**, we compared the roles of individual EP receptors in males and **females** . We found that the relative contribution of each EP-receptor subclass was

strikingly different in males from that in **females**. In **females**, the EP2 and EP4 receptors, which signal by stimulating adenylate cyclase, mediate the major portion of the vasodepressor **response** to PGE2. In males, the EP2 receptor has a modest effect, but most of the vasodepressor effect is mediated by the phospholipase C-coupled EP1 receptor. Finally in male mice, the EP3 receptor actively opposes the vasodepressor actions of **PGE**(2). Thus the hemodynamic actions of **PGE**(2) are mediated through complex interactions of several EP-receptor subtypes, and the role of individual EP receptors differs dramatically in males from that in **females**. These differences may contribute to **sexual** dimorphism of blood pressure regulation.

AB To identify the E-prostanoid (EP) receptors that mediate the hemodynamic actions of **PGE**(2), we studied acute vascular **responses** to infusions of **PGE**(2) using lines of mice in which each of four EP receptors (EP1 through EP4) have been disrupted by gene targeting. In mixed groups of males and **females**, vasodepressor **responses** after infusions of **PGE**(2) were significantly diminished in the EP2 (-/-) and EP4 (-/-) lines but not in the EP1 -/- or EP3 -/- lines. Because the actions of other hormonal systems that regulate blood pressure differ between **sexes**, we compared the roles of individual EP receptors in males and **females** . We found that the relative contribution of each EP-receptor subclass was

strikingly different in males from that in **females**. In **females**, the EP2 and EP4 receptors, which signal by stimulating adenylate cyclase, mediate the major portion of the vasodepressor **response** to PGE2. In males, the EP2 receptor has a modest effect, but most of the vasodepressor effect is mediated by the phospholipase C-coupled EP1 receptor. Finally in male mice, the EP3 receptor actively opposes the vasodepressor actions of **PGE**(2). Thus the hemodynamic actions of **PGE**(2) are mediated through complex interactions of several EP-receptor subtypes, and the role of individual EP receptors differs dramatically in males from that in **females**. These differences may contribute to **sexual** dimorphism of blood pressure regulation.

L25 ANSWER 5 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:664971 SCISEARCH
THE GENUINE ARTICLE: 229BM
TITLE: Glial-neuronal interactions in the neuroendocrine control of mammalian puberty: Facilitatory effects of gonadal steroids
AUTHOR: Ojeda S R (Reprint); Ma Y J
CORPORATE SOURCE: OREGON HLTH SCI UNIV, DIV NEUROSCI, OREGON REG PRIMATE RES
CTR, 505 NW 185TH AVE, BEAVERTON, OR 97006 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF NEUROBIOLOGY, (15 SEP 1999) Vol. 40, No. 4, pp.
528-540.
Publisher: JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK,
NY 10158-0012.
ISSN: 0022-3034.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 95
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB It is now clear that astroglial cells actively contribute to both the generation and how of information within the central nervous system, In the hypothalamus, astrocytes regulate the secretory activity of neuroendocrine neurons, A small subset of these neurons secrete luteinizing hormone-releasing hormone (LHRH), a neuropeptide essential for **sexual** development and adult reproductive function, Astrocytes stimulate LHRH secretion via cell-cell signaling mechanisms involving growth factors recognized by receptors with either serine/threonine or tyrosine kinase activity. Two members of the epidermal growth factor (EGF) family and their respective tyrosine kinase receptors appear to play key roles in this regulatory process. Transforming growth factor-alpha (TGF alpha) and its distant congeners, the neuregulins (NRGs), are produced in hypothalamic astrocytes, They stimulate LHRH secretion indirectly, via activation of erbB-1/erbB-2 and erbB-4/erbB-2 receptor complexes also located on astrocytes, Activation of these receptors leads to release of prostaglandin E-2 (**PGE**(2)), which then binds to specific receptors on LHRH neurons to elicit LHRH secretion. Gonadal steroids facilitate this glia-to-neuron communication process by acting at three different steps along the signaling pathway, They (a) increase astrocytic gene expression of at least one of the EGF-related ligands (TGF alpha), (b) increase expression of at least two of the receptors (erbB-4 and erbB-2), and (c) enhance the LHRH **response** to **PGE**(2) by up-regulating in LHRH neurons the expression of specific **PGE** (2) receptor isoforms. Focal overexpression of TGF alpha in either the median eminence or preoptic area of the hypothalamus accelerates puberty, Conversely, blockade of either TGF alpha or NRG hypothalamic actions delays the process. Thus, both TGF alpha and NRGs appear to be physiological components of the central neuroendocrine mechanism controlling the initiation of **female** puberty. By facilitating growth factor signaling pathways in the hypothalamus, ovarian steroids accelerate the pace and progression of the pubertal process. (C) 1999 John Wiley & Sons, Inc.

AB . . . secretory activity of neuroendocrine neurons, A small subset of these neurons secrete luteinizing hormone-releasing hormone (LHRH), a neuropeptide essential for **sexual** development and adult reproductive function, Astrocytes stimulate LHRH secretion via cell-cell signaling mechanisms involving growth factors recognized by receptors with. . . of erbB-1/erbB-2 and erbB-4/erbB-2 receptor complexes also located on astrocytes, Activation of these receptors leads to release of prostaglandin E-2 (**PGE**(2)), which then binds to specific receptors on LHRH neurons to elicit LHRH secretion. Gonadal steroids facilitate this glia-to-neuron communication process. . . (TGF alpha), (b) increase expression of at least two of the receptors (erbB-4 and erbB-2), and (c) enhance the LHRH **response** to **PGE**(2) by up-regulating in LHRH neurons the expression of specific **PGE** (2) receptor isoforms. Focal overexpression of TGF alpha in either the median eminence or preoptic area of the hypothalamus accelerates puberty,. . . Thus, both TGF alpha and NRGs appear to be physiological components of the central neuroendocrine mechanism controlling the initiation of **female** puberty. By facilitating growth factor signaling pathways in the hypothalamus, ovarian steroids accelerate the pace and progression of the pubertal. . .

=> d 125 6-10 ibib abs

L25 ANSWER 6 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:412549 SCISEARCH
THE GENUINE ARTICLE: 199MZ
TITLE: Cytokines inhibit sexual behavior in female rats: II. Prostaglandins mediate the suppressive effects of interleukin-1 beta
AUTHOR: Avitsur R (Reprint); Weidenfeld J; Yirmiya R
CORPORATE SOURCE: HEBREW UNIV JERUSALEM, DEPT PSYCHOL, MT SCOPUS, IL-91905 JERUSALEM, ISRAEL (Reprint); HADASSAH UNIV HOSP, DEPT NEUROL, IL-91120 JERUSALEM, ISRAEL
COUNTRY OF AUTHOR: ISRAEL
SOURCE: BRAIN BEHAVIOR AND IMMUNITY, (MAR 1999) Vol. 13, No. 1, pp. 33-45.
Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0889-1591.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 57

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The proinflammatory cytokine interleukin-1 (IL-1) induces several behavioral alterations that are characteristic of illness, such as anorexia and reduced locomotor and social activity. We have recently demonstrated that IL-1 inhibits **sexual** activity, motivation and attractivity in **female**, but not in male rats following either central or peripheral administration. Tn the present study we examined the involvement of prostaglandin (PG) synthesis in mediating IL-1-induced suppression of **female sexual** behavior. Administration of the cyclooxygenase blockers indomethacin or ibuprofen completely prevented IL-1-induced suppression of **female sexual** behavior, including the reduction in preceptive behavior, the lordosis **response** to a male's mounts, and the preference for a **sexually** active partner. In a subsequent study, ex-vivo release of hypothalamic **PGE**(2) and the secretion of corticosterone (CS) were measured in males and estrous **females** following IL-1 administration. At the same time and dose of IL-1 administration that significantly reduced **sexual** behavior in **female** but not male rats, IL-1 produced a significant increase in **PGE**(2) release in **female**, but not in male rats. In contrast, IL-1 induced a significant elevation of serum CS levels in males but not in **females**. These findings suggest that PG synthesis is involved in mediating the effects of IL-1 on **female sexual** behavior. Furthermore, differential secretion of PGs and CS may underlie the gender difference in the effects of IL-1 on **sexual** behavior. (C) 1999 Academic Press.

L25 ANSWER 7 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:219564 SCISEARCH
THE GENUINE ARTICLE: 175KM
TITLE: Seminal plasma components stimulate interleukin-8 and interleukin-10 release
AUTHOR: Denison F C (Reprint); Grant V E; Calder A A; Kelly R W
CORPORATE SOURCE: UNIV EDINBURGH, DEPT OBSTET & GYNAECOL, CTR REPROD BIOL, 37 CHALMERS ST, EDINBURGH EH3 9ET, MIDLOTHIAN, SCOTLAND (Reprint); UNIV EDINBURGH, MRC, CTR REPROD BIOL, REPROD BIOL UNIT, EDINBURGH EH3 9ET, MIDLOTHIAN, SCOTLAND
COUNTRY OF AUTHOR: SCOTLAND
SOURCE: MOLECULAR HUMAN REPRODUCTION, (MAR 1999) Vol. 5, No. 3,

pp. 220-226.
Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND.
ISSN: 1360-9947.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 40

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Human seminal plasma has potent anti-inflammatory properties which are thought to confer a survival advantage to the spermatozoa within the hostile **female genital** tract. In contrast, a profound pro-inflammatory leukocytosis has been observed post-coitus in animals and humans. Whether components of seminal plasma are involved in initiating this leukocytic reaction is not known. This study investigated the effect of human seminal plasma, a seminal plasma fraction and its principal constituent prostaglandins, prostaglandin E-2 (**PGE**(2)) and 19-hydroxy **PGE**, on the release of the pro-inflammatory neutrophil chemotactic factor interleukin-8 (IL-8) and the anti-inflammatory cytokines interleukin-10 (IL-10) and secretory leukocyte protease inhibitor (SLPI). The tissues studied were non-pregnant cervical explants, peripheral blood and the monocyte cell line U937. Seminal plasma fraction (SPF) significantly ($P < 0.05$) stimulated release of IL-8 and inhibited release of SLPI from non-pregnant cervical explants. SPF, PGE2 and 19-hydroxy **PGE** significantly ($P < 0.005$) stimulated IL-8 release from peripheral blood and U937 cells. 19-hydroxy **PGE** was significantly ($P < 0.005$) more effective than PGE2 in stimulating IL-8 release. Seminal plasma, SPF and **PGE**, significantly ($P < 0.05$) stimulated IL-10 release from U937 cells. 19-hydroxy **PGE** stimulated IL-10 release from U937 cells but this failed to reach significance. Release of IL-10 by cervical explants and SLPI by peripheral blood and U937 cells were below the detection limit of the assays employed. We suggest that the anti- and pro-inflammatory immune **responses** which seminal plasma induces might act in combination initially to promote sperm survival and then to facilitate their removal from the **female genital** tract.

L25 ANSWER 8 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1998:939912 SCISEARCH
THE GENUINE ARTICLE: BL98W
TITLE: Vasopressin-induced antipyresis - Sex- and experience-dependent febrile responses
AUTHOR: Pittman Q J (Reprint); Chen X H; Mouihate A; Martin S
CORPORATE SOURCE: UNIV CALGARY, HLTH SCI CTR, DEPT PHYSIOL & BIOPHYS, FAC MED, 3330 HOSP DR NW, CALGARY, AB T2N 4N1, CANADA (Reprint); UNIV CALGARY, FAC MED, NEUROSCI RES GRP, CALGARY, AB T2N 4N1, CANADA; MT ST VINCENT UNIV, DEPT BIOL, HALIFAX, NS B3M 2J6, CANADA
COUNTRY OF AUTHOR: CANADA
SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (NOV 1998) Vol. 856, pp. 53-61.
Publisher: NEW YORK ACAD SCIENCES, 2 EAST 63RD ST, NEW YORK, NY 10021.
ISSN: 0077-8923.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE

LANGUAGE: English
REFERENCE COUNT: 45
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB There is now good evidence that vasopressin (AVP) acts, in the mate rat, as a neurotransmitter in the ventral septal area to reduce fever. In light of the well. known **sexual** dimorphism in the AVP innervation of the brain, we asked if **female** rats would (a) display fevers different from those seen in mate rats, (b) **respond** to AVP with antipyresis, (c) display evidence of endogenous AVP-induced antipyresis during fever, and (d) display altered fevers and AVP involvement as a function of hormonal status. Oar experiments indicate that **female** rats display larger fevers to intracranial prostaglandin E-2 (**PGE**(2)) but not to systemic lipopolysaccharide or interleukin-1 beta than do male rats. The larger fevers may be due, in part, to a lack of AVP-induced antipyresis, as an AVP antagonist elevates **PGE**(2) fever in male but not in **female** rats and dialysates of the ventral septal area show increased AVP levels only in male rats during defervescence. Nonetheless, **females respond** to exogenous AVP with antipyresis. Throughout late pregnancy, parturition, and lactation, **PGE**(2) fevers are reduced, but this appears to be due to a general suppression of autonomic output not involving enhanced AVP antipyresis. Fevers due to lipopolysaccharide and interleukin-1 beta are also suppressed at this time, and in some animals, fevers are dramatically suppressed at about the time of parturition. Our results indicate that **female** rats may utilize different strategies for antipyresis than do male rats and that hormonal status may influence both peripherally generated and centrally activated fevers.

L25 ANSWER 9 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1998:113935 SCISEARCH
THE GENUINE ARTICLE: BK30S
TITLE: Aromatase expression in health and disease
AUTHOR: Simpson E R (Reprint); Zhao Y; Agarwal V R; Michael M D; Bulun S E; Hinshelwood M M; GrahamLorence S; Sun T J; Fisher C R; Qin K N; Mendelson C R
CORPORATE SOURCE: UNIV TEXAS, SW MED CTR, CECIL H & IDA GREEN CTR REPROD BIOL SCI, DALLAS, TX 75235 (Reprint); UNIV TEXAS, SW MED CTR, DEPT OBSTET GYNECOL, DALLAS, TX 75235; UNIV TEXAS, SW MED CTR, DEPT BIOCHEM, DALLAS, TX 75235
COUNTRY OF AUTHOR: USA
SOURCE: RECENT PROGRESS IN HORMONE RESEARCH, (JAN 1997) Vol. 52, pp. 185-214. Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE 500, BETHESDA, MD 20814-4410. ISSN: 0079-9963.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 123
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Family 19 of the P450 superfamily is **responsible** for the conversion of C-19 androgenic steroids to the corresponding estrogens, a reaction known as aromatization, since it involves conversion of the Delta(4)-3-one A-ring of the androgens to the corresponding phenolic A-ring characteristic of estrogens. Its members occur throughout the entire vertebrate phylum. The reaction mechanism of aromatase is very interesting from a chemical point of view and has been studied

extensively; however, a detailed examination of structure-function relationships has not been possible due to lack of a crystal structure. Recent attempts to model the three-dimensional structure of aromatase have permitted a model that accounts for the reaction mechanism and predicts the location of aromatase inhibitors. The gene encoding human aromatase has been cloned and characterized and shown to be unusual compared to genes encoding other P450 enzymes, since there are a number of untranslated first exons that occur in aromatase transcripts in a tissue-specific fashion, due to differential splicing as a consequence of the use of tissue-specific promoters. Thus, expression in ovary utilizes a proximal promoter that is regulated primarily by cAMP. On the other hand, expression in placenta utilizes a distal promoter that is located at least 40 kb upstream of the start of transcription and that is regulated by retinoids. Other promoters are employed in brain and adipose tissue. In the latter case, class 1 cytokines such as IL-6 and IL-11 as well as TNF alpha are important regulatory factors. **PGE**(2) is also an important regulator of aromatase expression in adipose mesenchymal cells via cAMP and **PGE**(2) appears to be a major factor produced by breast tumors that stimulates estrogen biosynthesis in local mesenchymal sites. In all of the splicing events involved in the use of these various promoters, a common 3'-splice junction is employed that is located upstream of the start of translation; thus, the coding regions of the transcripts-and hence the protein-are identical regardless of the tissue site of expression; what differ in a tissue-specific fashion are the 5'-ends of the transcripts. This pattern of expression has great significance both from a phylogenetic and ontogenetic standpoint as well as for the physiology and pathophysiology of estrogen formation. Recently, a number of mutations of the aromatase gene have been described, which give rise to complete estrogen deficiency. In **females** this results in virilization in utero and primary amenorrhea with hypergonadotropic hypogonadism at the time of puberty. In men the most striking feature is continued linear bone growth beyond the time of puberty, delayed bone age, and failure of epiphyseal closure, thus indicating an important role of estrogens in bone metabolism in men. In both **sexes** the symptoms can be alleviated by estrogen administration.

L25 ANSWER 10 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 97:868473 SCISEARCH
THE GENUINE ARTICLE: YG185
TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women
AUTHOR: Farker K (Reprint); Schweer H; Vollandt R; Nassr N; Nagel U; Seyberth H W; Hoffmann A; Oettel M
CORPORATE SOURCE: UNIV JENA, INST CLIN PHARMACOL, D-07740 JENA, GERMANY (Reprint); UNIV MARBURG, DEPT PEDIAT, D-35033 MARBURG, GERMANY; UNIV JENA, INST MED STAT INFORMAT & DOCUMENTAT, D-07740 JENA, GERMANY; JENAPHARM GMBH, D-07745 JENA, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: PROSTAGLANDINS, (SEP 1997) Vol. 54, No. 3, pp. 655-664. Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010. ISSN: 0090-6980.
DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 18
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E-2 (**PGE** (2)), 6-keto-PGF(1 alpha), thromboxane B-2 (TxB(2)) and their metabolites **PGE**-M (11 alpha-hydroxy-9,15-dioxo-2,3,4,5,20-pentanor-19-carboxyprostanoic acid), 2,3-dinor-6-keto-PGF(1 alpha), 2,3-dinor-TxB(2) and 11-dehydro-TxB(2) were determined by gas chromatography-triple stage quadrupole mass spectrometry (GC/MS/MS) in 41 young nonpregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women**. Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, **PGE**(2) and TxB(2) were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels ni estradiol and progesterone were different in young and post-menopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins. Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

=> s l25 range=,1998
'CN' IS NOT A VALID FIELD CODE
'CN' IS NOT A VALID FIELD CODE
4 FILES SEARCHED...
'CN' IS NOT A VALID FIELD CODE
5 FILES SEARCHED...
'CN' IS NOT A VALID FIELD CODE
8 FILES SEARCHED...
12 FILES SEARCHED...
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L65 (S) L66'
L29 36 L25

=> d ibib abs 1-10

L29 ANSWER 1 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1998:939912 SCISEARCH
THE GENUINE ARTICLE: BL98W
TITLE: Vasopressin-induced antipyresis - Sex- and experience-dependent febrile responses
AUTHOR: Pittman Q J (Reprint); Chen X H; Mouihate A; Martin S
CORPORATE SOURCE: UNIV CALGARY, HLTH SCI CTR, DEPT PHYSIOL & BIOPHYS, FAC MED, 3330 HOSP DR NW, CALGARY, AB T2N 4N1, CANADA (Reprint); UNIV CALGARY, FAC MED, NEUROSCI RES GRP, CALGARY, AB T2N 4N1, CANADA; MT ST VINCENT UNIV, DEPT BIOL, HALIFAX, NS B3M 2J6, CANADA
COUNTRY OF AUTHOR: CANADA
SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (NOV 1998) Vol. 856, pp. 53-61.

Publisher: NEW YORK ACAD SCIENCES, 2 EAST 63RD ST, NEW YORK, NY 10021.
ISSN: 0077-8923.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 45
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB There is now good evidence that vasopressin (AVP) acts, in the mate rat, as a neurotransmitter in the ventral septal area to reduce fever. In light of the well. known **sexual** dimorphism in the AVP innervation of the brain, we asked if **female** rats would (a) display fevers different from those seen in mate rats, (b) **respond** to AVP with antipyresis, (c) display evidence of endogenous AVP-induced antipyresis during fever, and (d) display altered fevers and AVP involvement as a function of hormonal status. Oar experiments indicate that **female** rats display larger fevers to intracranial prostaglandin E-2 (**PGE**(2)) but not to systemic lipopolysaccharide or interleukin-1 beta than do male rats. The larger fevers may be due, in part, to a lack of AVP-induced antipyresis, as an AVP antagonist elevates **PGE**(2) fever in male but not in **female** rats and dialysates of the ventral septal area show increased AVP levels only in male rats during defervescence. Nonetheless, **females respond** to exogenous AVP with antipyresis. Throughout late pregnancy, parturition, and lactation, **PGE**(2) fevers are reduced, but this appears to be due to a general suppression of autonomic output not involving enhanced AVP antipyresis. Fevers due to lipopolysaccharide and interleukin-1 beta are also suppressed at this time, and in some animals, fevers are dramatically suppressed at about the time of parturition. Our results indicate that **female** rats may utilize different strategies for antipyresis than do male rats and that hormonal status may influence both peripherally generated and centrally activated fevers.

L29 ANSWER 2 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1998:113935 SCISEARCH
THE GENUINE ARTICLE: BK30S
TITLE: Aromatase expression in health and disease
AUTHOR: Simpson E R (Reprint); Zhao Y; Agarwal V R; Michael M D; Bulun S E; Hinshelwood M M; GrahamLorence S; Sun T J; Fisher C R; Qin K N; Mendelson C R
CORPORATE SOURCE: UNIV TEXAS, SW MED CTR, CECIL H & IDA GREEN CTR REPROD BIOL SCI, DALLAS, TX 75235 (Reprint); UNIV TEXAS, SW MED CTR, DEPT OBSTET GYNECOL, DALLAS, TX 75235; UNIV TEXAS, SW MED CTR, DEPT BIOCHEM, DALLAS, TX 75235
COUNTRY OF AUTHOR: USA
SOURCE: RECENT PROGRESS IN HORMONE RESEARCH, (JAN 1997) Vol. 52, pp. 185-214.
Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE 500, BETHESDA, MD 20814-4410.
ISSN: 0079-9963.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 123
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Family 19 of the P450 superfamily is **responsible** for the conversion of C-19 androgenic steroids to the corresponding estrogens, a

reaction known as aromatization, since it involves conversion of the Delta(4)-3-one A-ring of the androgens to the corresponding phenolic A-ring characteristic of estrogens. Its members occur throughout the entire vertebrate phylum. The reaction mechanism of aromatase is very interesting from a chemical point of view and has been studied extensively; however, a detailed examination of structure-function relationships has not been possible due to lack of a crystal structure. Recent attempts to model the three-dimensional structure of aromatase have permitted a model that accounts for the reaction mechanism and predicts the location of aromatase inhibitors. The gene encoding human aromatase has been cloned and characterized and shown to be unusual compared to genes encoding other P450 enzymes, since there are a number of untranslated first exons that occur in aromatase transcripts in a tissue-specific fashion, due to differential splicing as a consequence of the use of tissue-specific promoters. Thus, expression in ovary utilizes a proximal promoter that is regulated primarily by cAMP. On the other hand, expression in placenta utilizes a distal promoter that is located at least 40 kb upstream of the start of transcription and that is regulated by retinoids. Other promoters are employed in brain and adipose tissue. In the latter case, class 1 cytokines such as IL-6 and IL-11 as well as TNF alpha are important regulatory factors. **PGE**(2) is also an important regulator of aromatase expression in adipose mesenchymal cells via cAMP and **PGE**(2) appears to be a major factor produced by breast tumors that stimulates estrogen biosynthesis in local mesenchymal sites. In all of the splicing events involved in the use of these various promoters, a common 3'-splice junction is employed that is located upstream of the start of translation; thus, the coding regions of the transcripts-and hence the protein-are identical regardless of the tissue site of expression; what differ in a tissue-specific fashion are the 5'-ends of the transcripts. This pattern of expression has great significance both from a phylogenetic and ontogenetic standpoint as well as for the physiology and pathophysiology of estrogen formation. Recently, a number of mutations of the aromatase gene have been described, which give rise to complete estrogen deficiency. In **females** this results in virilization in utero and primary amenorrhea with hypergonadotropic hypogonadism at the time of puberty. In men the most striking feature is continued linear bone growth beyond the time of puberty, delayed bone age, and failure of epiphyseal closure, thus indicating an important role of estrogens in bone metabolism in men. In both **sexes** the symptoms can be alleviated by estrogen administration.

L29 ANSWER 3 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 97:868473 SCISEARCH
THE GENUINE ARTICLE: YG185
TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women
AUTHOR: Farker K (Reprint); Schweer H; Vollandt R; Nassr N; Nagel U; Seyberth H W; Hoffmann A; Oettel M
CORPORATE SOURCE: UNIV JENA, INST CLIN PHARMACOL, D-07740 JENA, GERMANY (Reprint); UNIV MARBURG, DEPT PEDIAT, D-35033 MARBURG, GERMANY; UNIV JENA, INST MED STAT INFORMAT & DOCUMENTAT, D-07740 JENA, GERMANY; JENAPHARM GMBH, D-07745 JENA, GERMANY
COUNTRY OF AUTHOR: GERMANY

SOURCE: PROSTAGLANDINS, (SEP 1997) Vol. 54, No. 3, pp. 655-664.
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010.
ISSN: 0090-6980.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 18
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E-2 (**PGE** (2)), 6-keto-PGF(1 alpha), thromboxane B-2 (TxB(2)) and their metabolites **PGE**-M (11 alpha-hydroxy-9,15-dioxo-2,3,4,5,20-pentanor-19-carboxyprostanoic acid), 2,3-dinor-6-keto-PGF(1 alpha), 2,3-dinor-TxB(2) and 11-dehydro-TxB(2) were determined by gas chromatography-triple stage quadrupole mass spectrometry (GC/MS/MS) in 41 young nonpregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women**. Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, **PGE**(2) and TxB(2) were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels ni estradiol and progesterone were different in young and post-menopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins. Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

L29 ANSWER 4 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 97:647831 SCISEARCH
THE GENUINE ARTICLE: XT349
TITLE: Enhancement by sex hormones of the osteoregulatory effects of mechanical loading and prostaglandins in explants of rat ulnae
AUTHOR: Cheng M Z; Zaman G; Rawlinson S C F; Pitsillides A A; Suswillo R F L; Lanyon L E (Reprint)
CORPORATE SOURCE: UNIV LONDON ROYAL VET COLL, ROYAL COLL ST, LONDON NW1 0TU, ENGLAND (Reprint); UNIV LONDON ROYAL VET COLL, LONDON NW1 0TU, ENGLAND
COUNTRY OF AUTHOR: ENGLAND
SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (SEP 1997) Vol. 12, No. 9, pp. 1424-1430.
Publisher: BLACKWELL SCIENCE INC, 350 MAIN ST, MALDEN, MA 02148.
ISSN: 0884-0431.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 41
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Explants of ulnae from 5-week-old male and **female** rats were

cleaned of marrow and soft tissue and, in the presence and absence of 10(-8) M 17 beta-estradiol (E2) or 5 alpha-dihydrotestosterone (DHT), mechanically loaded or treated with exogenous prostanoids previously shown to be produced during loading. Over an 18-h period, mechanical loading (peak strain 1300 mu epsilon, 1 Hz, 8 minutes, maximum strain rate 25,000 mu epsilon/s), prostaglandin E-2 (**PGE**(2)) and prostacyclin (PGI(2)) (10(-6) M), each separately produced quantitatively similar increases in cell proliferation and matrix production in bones from males and **females**, as indicated by incorporation of [H-3]thymidine into DNA and [H-3]proline into collagen. E2 and DHT both increased [H-3]thymidine and [H-3]proline incorporations, E2 producing greater increases in **females** than in males. Indomethacin abrogated the effects of loading, but had no effects on those of **sex** hormones. Loading, or prostanoids, together with **sex** hormones, produced **responses** generally equal to or greater than the addition of the individual influences acting independently. In **females** there was a synergistic **response** in [H-3] thymidine incorporation between loading and E2, which was quantitatively similar to the interaction between E2 and **PGE**(2) or PGI(2). The interaction between loading and E-2 for [H-3]proline incorporation was not mimicked by these prostanoids. In males the synergism in [H-3]proline incorporation seen between loading and DHT was mimicked by that between PGI(2) and DHT. We conclude that loading stimulates increased bone cell proliferation and matrix production in situ through a prostanoid-dependent mechanism. This **response** is equal in size in males and **females**. Estrogen and testosterone increase proliferation and matrix production through a mechanism independent of prostanoid production. The interactions between loading and hormones are reproduced in some but not all cases by E2 and prostaglandins. E2 with loading and prostaglandins has greater effects in **female** bones, while DHT with loading and prostaglandins has greater effects in males.

L29 ANSWER 5 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 97:387605 SCISEARCH
THE GENUINE ARTICLE: WY547
TITLE: A cytokine switch induced by human seminal plasma: An immune modulation with implications for sexually transmitted disease
AUTHOR: Kelly R W (Reprint); Carr G G; Critchley H O D
CORPORATE SOURCE: UNIV EDINBURGH, CTR REPROD BIOL, MRC, REPROD BIOL UNIT, 37 CHALMERS ST, EDINBURGH EH3 9EW, MIDLOTHIAN, SCOTLAND (Reprint); UNIV EDINBURGH, CTR REPROD BIOL, DEPT OBSTET & GYNAECOL, EDINBURGH EH3 9EW, MIDLOTHIAN, SCOTLAND
COUNTRY OF AUTHOR: SCOTLAND
SOURCE: HUMAN REPRODUCTION, (APR 1997) Vol. 12, No. 4, pp. 677-681
Publisher: OXFORD UNIV PRESS, WALTON ST JOURNALS DEPT, OXFORD, ENGLAND OX2 6DP.
ISSN: 0268-1161.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 36

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The immunosuppressive activity of human seminal plasma may be one factor in the aetiology of **sexually** transmitted disease and could be particularly important for the spread of human immunodeficiency

virus (HIV), The advent of virus that can preferentially infect Langerhans cells of the **genital** mucosa underscores the relevance of seminal plasma effects, Virally infected cells are eradicated by the killing activity of T cells and natural killer (NK) cells and this cytotoxicity is stimulated by IL-12 (previously known as natural killer cell stimulatory factor) and partly inhibited by IL-10 (previously known as cytokine synthesis inhibitory factor), We have examined the effects of human seminal plasma on the production of these key cytokines, Cytokine production was measured in rapidly diluted, fresh, lipopolysaccharide (LPS)-stimulated, whole blood since this provided leukocytes with minimal exposure to prostaglandin, Prostaglandin concentrations and cytokine release were measured by ELISA, Addition of human seminal plasma diluted up to 100 000 times (0.001%) to blood cell cultures led to a marked increase in the IL-10/IL-12 ratio (P <0.02), A dose-dependent increase in the ratio was observed in five separate experiments, from a control value of 1 (no seminal plasma) to a mean value of 80 (1% seminal plasma), This cytokine switch was also seen when seminal plasma was substituted by pure prostaglandin E (**PGE**) and 19-OH **PGE** (the main prostaglandin constituent of human seminal plasma), Lipid-extracted seminal plasma was considerably less active at high dilutions than whole seminal plasma at the same dilution, However, its activity could be restored by the addition of synthetic **PGE** and 19-hydroxy **PGE**, A stimulation of IL-10 and a decrease in IL-12 in host-defence cells of the lower **female**-reproductive tract will seriously affect the ability of cytotoxic T cells and NK cells to recognise and destroy virally infected cells, In addition, the stimulation of IL-10 will inhibit the release of the anti-HIV activity from CD8+ve cells, The cytokine switch reported here, activated by semen deposition, would exercise a key inhibitory control over vital immune defences in the lower **genital** tract, with ablation of cell-mediated **responses** and immunosurveillance. (C) European Society for Human Reproduction and Embryology.

L29 ANSWER 6 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 97:386214 SCISEARCH
THE GENUINE ARTICLE: WY626
TITLE: Differential ventral septal vasopressin release is associated with sexual dimorphism in PGE(2) fever
AUTHOR: Chen X (Reprint); Landgraf R; Pittman Q J
CORPORATE SOURCE: UNIV CALGARY, DEPT PHYSIOL & BIOPHYS, NEUROSCI RES GRP, 3330 HOSP DR NW, CALGARY, AB T2N 4N1, CANADA (Reprint); MAX PLANCK INST PSYCHIAT, INST CLIN, D-80804 MUNICH, GERMANY
COUNTRY OF AUTHOR: CANADA; GERMANY
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-REGULATORY INTEGRATIVE AND COMPARATIVE PHYSIOLOGY, (MAY 1997) Vol. 41, No. 5, pp. R1664-R1669.
Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 0363-6119.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 30
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The vasopressinergic innervation of the ventral septal area (VSA) has been shown to be implicated in antipyresis. Because this system is less

well developed in **female** rats, we hypothesized that **female** rats would display exaggerated febrile **responses**. We therefore examined the temperature **responses** of conscious and urethan-anesthetized rats of both **sexes** to centrally administered prostaglandin E-2 (**PGE**(2)) and correlated these **responses** with the release and action of endogenous arginine vasopressin (AVP) in the VSA. Both conscious [25 ng/5 mu l **PGE**(2) intracerebroventricularly (icv)] and anesthetized (VSA microdialyzed, 50 ng/5 mu l **PGE**(2) icv) **female** rats had higher fevers than did males. Infusion of an AVP V-1a receptor antagonist [1 nmol [d(CH2)(5)Tyr(Me)]AVP] plus **PGE**(2) gave rise to higher fevers in males but not in **females**. Measurements of AVP in microdialysates of the VSA showed that the release of endogenous AVP was increased in **response** to **PGE**(2) in males only. Baseline AVP release in both **sexes** was similar. The results suggest that there is a **sex**-related difference in **PGE**(2) fever, which may be accounted for by the differential AVP release in the VSA.

L29 ANSWER 7 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 96:916161 SCISEARCH
THE GENUINE ARTICLE: VW830
TITLE: Self-reported sexual function and sexual arousability in women with epilepsy
AUTHOR: Morrell M J (Reprint); Guldner G T
CORPORATE SOURCE: STANFORD UNIV, MED CTR, DEPT NEUROL & NEUROL SCI, SCH MED,
STANFORD COMPREHENS EPILEPSY CTR, H3160, STANFORD, CA 94307 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: EPILEPSIA, (DEC 1996) Vol. 37, No. 12, pp. 1204-1210.
Publisher: LIPPINCOTT-RAVEN PUBL, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106.
ISSN: 0013-9580.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 35

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Purpose: Women with epilepsy are at risk for sexual dysfunction but the frequency and types of dysfunction have not been well characterized.

Methods: Self-reported sexual function was evaluated in 116 women aged 18-65 years with epilepsy and no concomitant medical or psychiatric illness, including 99 with localization-related epilepsy (LRE) and 17 with primary generalized epilepsy (PGE). Variables evaluated included seizure frequency, age of seizure onset, and antiepileptic drug (AED) exposure. Standardized inventories assessed sexual functioning, sexual arousability and anxiety, sexual behavior, and depression.

Results: Although **sexual** experience was not reduced, **women** with **PGE** and LRE reported significantly less **sexual** arousability and **women** with LRE reported significantly more **sexual** anxiety. **Women** with LRE experienced significantly more dyspareunia, vaginismus, **arousal** insufficiency, and **sexual** dissatisfaction, whereas **women** with **PGE** experienced anorgasmia and **sexual** dissatisfaction. **Sexual** symptoms were not associated with seizure frequency, AED exposure, **sexual** experience, depression, or prepubertal seizure onset.

Conclusions: In contrast to subjects of previous research, the women in

our study did not have a disorder of sexual desire, but more than one third experienced disorders of sexual arousal, implying a physiological deficit. Although the etiology for these arousal phase dysfunctions has not been defined, such conditions are treatable and warrant referral to a gynecologist versed in the treatment of sexual disorders.

L29 ANSWER 8 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 96:479396 SCISEARCH
THE GENUINE ARTICLE: UT138
TITLE: PROGESTERONE AND DEXAMETHASONE STIMULATE PROLIFERATION AND
DIFFERENTIATION OF OSTEOPROGENITORS AND PROGENITORS FOR ADIPOCYTES AND MACROPHAGES IN CELL-POPULATIONS DERIVED FROM ADULT-RAT VERTEBRAE
AUTHOR: ISHIDA Y (Reprint); TERTINEGG I; HEERSCHE J N M
CORPORATE SOURCE: UNIV TORONTO, FAC DENT, ROOM 400, 124 EDWARD ST, TORONTO, ON M5G 1G6, CANADA (Reprint)
COUNTRY OF AUTHOR: CANADA
SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (JUL 1996) Vol. 11, No. 7, pp. 921-930.
ISSN: 0884-0431.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 36

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We investigated the effects of the **sex** hormone progesterone (Prog) and the synthetic glucocorticoid dexamethasone (Dex) on proliferation and differentiation of progenitor cells of osteogenic, adipocytic, and hemopoietic lineages in cell populations derived from explants of adult **female** rat lumbar vertebrae. The cell populations were obtained by culturing bone explants in plasma clots immersed in a-minimum essential medium plus 10% fetal calf serum (standard medium) and then subculturing the outgrowth cells in standard medium plus 50 mu g/ml of ascorbic acid, 5 mM beta-glycerophosphate, and with or without Frog or Dex. On day 6 of culture, these populations were analyzed for cAMP **responses** to parathyroid hormone (PTH), prostaglandin E(2) (**PGE**(2)), and isoproterenol (IPT). Increases in intracellular cAMP were seen in **response** to PTH, **PGE** (2), and IPT, and culturing in medium containing Frog increased these **responses**. At various time periods between days 4-27 of culture, the cultures were evaluated for the presence of bone nodules, alkaline phosphatase (AP)-positive colonies, adipocytes, monocytes, and macrophages. Frog and Dex increased the number of bone nodules and AP-positive colonies. The effect of Frog on bone nodule formation was smaller than that of Dex. In addition, the effect of Dex on bone nodule formation was evident after 10 days of culture, while the Frog-induced effects became significant at days 16-20 of culture. Both hormones also increased the number of Sudan IV-positive colonies (adipocytes), certain types of alpha-naphthyl butyrate esterase (alpha-NBE)-positive colonies (monocytes, macrophages, and T-lymphocytes), and ED2-positive colonies (macrophages). Frog-treated cultures contained more colonies of small spindle-shaped alpha-NBE-positive cells and fewer colonies of small round alpha-NBE-positive cells when compared with Dex-treated cultures. These data indicate that cell populations derived from adult rat lumbar vertebrae contain, among others, osteoprogenitors and progenitors for adipocytes and macrophages that are stimulated to proliferate and differentiate by Frog and Dex. The data also suggest that the effects of frog and Dex differ qualitatively and quantitatively.

L29 ANSWER 9 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 95:80195 SCISEARCH
THE GENUINE ARTICLE: QB499
TITLE: MESENTERIC VASCULAR-RESPONSES TO VASOPRESSIN DURING DEVELOPMENT OF DOCA-SALT HYPERTENSION IN MALE AND FEMALE RATS
AUTHOR: STALLONE J N (Reprint)
CORPORATE SOURCE: NE OHIO UNIV, COLL MED, DEPT PHYSIOL, 4209 STATE RT 44, POB 95, ROOTSTOWN, OH, 44272 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-REGULATORY INTEGRATIVE AND COMPARATIVE PHYSIOLOGY, (JAN 1995) Vol. 37, No. 1, pp. R40-R49.
ISSN: 0363-6119.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 58

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Deoxycorticosterone acetate (DOCA)-salt hypertension develops to a greater extent in male (M) than in **female** (F) rats. To determine the role of the vasculature, reactivity to arginine vasopressin (AVP) and prostanoid output were examined in the isolated perfused mesenteric vasculature of hypertensive (HT) and normotensive-control (NTC) M and F rats after acute (1-wk) and chronic (4-wk) DOCA-salt treatment. Systolic blood pressure was significantly higher in M than in F HT rats (187 +/- 3 vs. 151 +/- 3 mmHg after 4 wk; P < 0.02). After acute treatment, vascular reactivity to AVP (maximal perfusion pressure) in HT was elevated in M (181 +/- 18 mmHg; P < 0.02) but not in F (135 +/- 6 mmHg) compared with NTC (90 +/- 6 mmHg, M vs. 119 +/- 5 mmHg, F). After chronic treatment, vascular reactivity to AVP in HT was elevated in both **sexes** (P < 0.02), although more in F (175 +/- 13 mmHg) than in M (141 +/- 11 mmHg). In contrast, vascular **responsiveness** to phenylephrine did not differ significantly between M and F NTC or HT preparations after either acute or chronic treatment. **Sex** differences in basal and AVP-induced 6-ketoprostaglandin (6-keto-PG) F-1 alpha and **PGE**(2) output by HT and NTC vasculature were reciprocal to **sex** differences in the vasoconstriction **responses** to AVP. After acute treatment, AVP-stimulated 6-keto-PGF(1 alpha) output by HT was elevated slightly in F (33.6 +/- 1.7 ng/3 min; P less than or equal to 0.02) but not in M (49.9 +/- 4.3 ng/3 min) compared with NTC (23.5 +/- 2.6 ng/3 min, F vs. 34.7 +/- 4.9 ng/3 min, M). After chronic treatment, output by HT was enhanced in both **sexes** (P less than or equal to 0.02), although more in M (109 +/- 15.4 ng/3 min) than in F (68 +/- 6.6 ng/3 min). These findings suggest that **sex** differences in the relative balance between AVP-induced vasoconstriction and vasodilatory prostanoid release may contribute to male-**female** differences in mesenteric vascular reactivity to AVP in NT and that disturbances in this balance may be **responsible**, at least in part, for the **sex**- and time-dependent changes in reactivity to AVP observed during the development of DOCA-salt hypertension.

L29 ANSWER 10 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 94:151690 SCISEARCH
THE GENUINE ARTICLE: MY446
TITLE: GENDER-RELATED RESPONSE TO A TERT-BUTYL HYDROPEROXIDE-INDUCED OXIDATION IN HUMAN NEONATAL TISSUE

AUTHOR: LAVOIE J C; CHESSEX P (Reprint)
CORPORATE SOURCE: UNIV MONTREAL, HOP STE JUSTINE, DEPT PEDIAT, RES CTR, 3175
CHEMIN COTE ST CATHERINE, MONTREAL H3T 1C5, PQ, CANADA (Reprint); UNIV MONTREAL, HOP STE JUSTINE, DEPT PEDIAT, RES CTR, MONTREAL H3T 1C5, PQ, CANADA; UNIV MONTREAL, HOP STE JUSTINE, DEPT PEDIAT, PERINATAL SERV, MONTREAL H3T 1C5, PQ, CANADA
COUNTRY OF AUTHOR: CANADA
SOURCE: FREE RADICAL BIOLOGY AND MEDICINE, (MAR 1994) Vol. 16, No. 3, pp. 307-313.
ISSN: 0891-5849.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 31

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Reports of gender-related differences in the activity of enzymes involved in the metabolism of intracellular antioxidants, led us to verify whether the prostaglandin **response** to tert-butyl hydroperoxide (TBH) differed according to the **sex** of infants. Segments of human umbilical veins were perfused in the presence or absence of TBH (0.25 mmol/l, and 1.0 mmol/l). Because TBH is quenched in the cell by glutathione peroxidase, total glutathione concentrations and the production of glutathione-dependent prostaglandins (**PGE**(2) and PCF2 alpha) as well as membrane-derived eicosanoids (PGI(2) and thromboxane) were measured in the eluate. In veins from boys, TBH induced a sustained **response** for glutathione only, which was increased ($p < 0.05$). In **female**-derived tissue, the hydroperoxide induced a different **response** according to the dose ofTBH. At 0.25 mmol/l, a drop ($p < 0.005$) in PGF(2) alpha was associated with a rise ($p < 0.001$) in thromboxane. At 1.0 mmol/l, TBH had an opposite effect-there was a rise ($p < 0.01$) in **PGE**(2) and PGI(2). The prostaglandin concentrations were not proportional to the oxidative stimulus, suggesting a critical level of TBH at which the oxidative state differes in tissues derived from boys or **girls**.

=> d ibib abs 11-20

L29 ANSWER 11 OF 36 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.
ACCESSION NUMBER: 1997-0518264 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Enhancement by sex hormones of the osteoregulatory effects of mechanical loading and prostaglandins in explants of rat ulnae
AUTHOR: MING ZHAO CHENG; ZAMAN G.; RAWLINSON S. C. F.; PITSILLIDES A. A.; SUSWILLO R. F. L.; LANYON L. E.
CORPORATE SOURCE: The Royal Veterinary College, University of London, London, United Kingdom
SOURCE: Journal of bone and mineral research, (1997), 12(9), 1424-1430, 42 refs.
ISSN: 0884-0431 CODEN: JBMREJ
DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-21114, 354000069214160120
AN 1997-0518264 PASCAL

AB Explants of ulnae from 5-week-old male and **female** rats were cleaned of marrow and soft tissue and, in the presence and absence of 10.sup.-.sup.8 M 17.beta.-estradiol (E2) or 5.alpha.-dihydrotestosterone (DHT), mechanically loaded or treated with exogenous prostanoids previously shown to be produced during loading. Over an 18-h period, mechanical loading (peak strain 1300 .mu..epsilon., I Hz, 8 minutes, maximum strain rate 25,000 .mu..epsilon./s), prostaglandin E.sub.2 (**PGE**.sub.2) and prostacyclin (PGI.sub.2) (10.sup.-.sup.6 M), each separately produced quantitatively similar increases in cell proliferation and matrix production in bones from males and **females**, as indicated by incorporation of [.sup.3H]thymidine into DNA and [.sup.3H]proline into collagen. E2 and DHT both increased [.sup.3H]thymidine and [.sup.3H]proline incorporations, E2 producing greater increases in **females** than in males. Indomethacin abrogated the effects of loading, but had no effects on those of **sex** hormones. Loading, or prostanoids, together with **sex** hormones, produced **responses** generally equal to or greater than the addition of the individual influences acting independently. In **females** there was a synergistic **response** in [.sup.3H]thymidine incorporation between loading and E2, which was quantitatively similar to the interaction between E2 and **PGE** .sub.2 or PGI.sub.2. The interaction between loading and E2 for [.sup.3H]proline incorporation was not mimicked by these prostanoids. In males the synergism in [.sup.3H]proline incorporation seen between loading and DHT was mimicked by that between PGI.sub.2 and DHT. We conclude that loading stimulates increased bone cell proliferation and matrix production in situ through a prostanoid-dependent mechanism. This **response** is equal in size in males and **females**. Estrogen and testosterone increase proliferation and matrix production through a mechanism independent of prostanoid production. The interactions between loading and hormones are reproduced in some but not all cases by E2 and prostaglandins. E2 with loading and prostaglandins has greater effects in **female** bones, while DHT with loading and prostaglandins has greater effects in males.

L29 ANSWER 12 OF 36 PASCAL
ACCESSION NUMBER: 1997-0326904 PASCAL

TITLE (IN ENGLISH): Differential ventral septal vasopressin release is associated with sexual dimorphism in PGE.sub.2 fever
AUTHOR: CHEN X.; LANDGRAF R.; PITTMAN Q. J.
CORPORATE SOURCE: Neuroscience Research Group, Department of Physiology and Biophysics, University of Calgary, Calgary, Alberta, T2N 4N1, Canada; Max Planck Institute of Psychiatry, Clinical Institute, 80804 Munich, Germany, Federal Republic of
SOURCE: American journal of physiology. Regulatory, integrative and comparative physiology, (1997), 41(5), R1664-R1669, 30 refs.
ISSN: 0363-6119 CODEN: AJPRDO
DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-670E, 354000065609740390
AN 1997-0326904 PASCAL
CP Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
AB The vasopressinergic innervation of the ventral septal area (VSA) has been shown to be implicated in antipyresis. Because this system is less well developed in **female** rats, we hypothesized that **female** rats would display exaggerated febrile **responses** . We therefore examined the temperature **responses** of conscious and urethan-anesthetized rats of both **sexes** to centrally administered prostaglandin E2 (PGE2) and correlated these **responses** with the release and action of endogenous arginine vasopressin (AVP) in the VSA. Both conscious [25 ng/5 .mu.l **PGE** .sub.2 intracerebroventricularly (icv)] and anesthetized (VSA microdialyzed, 50 ng/5 .mu.l PGE2 icv) **female** rats had higher fevers than did males. Infusion of an AVP Via receptor antagonist[1 nmol [d(CH.sub.2).sub.5TYr(Me)]AVP] plus **PGE**.sub.2 gave rise to higher fevers in males but not in **females**. Measurements of AVP in microdialysates of the VSA showed that the release of endogenous AVP was increased in **response** to **PGE**.sub.2 in males only. Baseline AVP release in both **sexes** was similar. The results suggest that there is a **sex**-related difference in PGE2 fever, which may be accounted for by the differential AVP release in the VSA.

L29 ANSWER 13 OF 36 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.
ACCESSION NUMBER: 1997-0307382 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): A cytokine switch induced by human seminal plasma : an immune modulation with implications for sexually transmitted disease
AUTHOR: KELLY R. W.; CARR G. G.; CRITCHLEY H. O. D.
CORPORATE SOURCE: Medical Research Council Reproductive Biology Unit, University of Edinburgh Centre for Reproductive Biology, 37 Chalmers Street, Edinburgh EH3 9EW, United Kingdom; Department of Obstetrics and Gynaecology, University of Edinburgh Centre for Reproductive Biology, 37 Chalmers Street, Edinburgh EH3 9EW, United Kingdom
SOURCE: Human reproduction : (Oxford), (1997), 12(4), 677-681,
36 refs.
ISSN: 0268-1161 CODEN: HUREEE
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United Kingdom
LANGUAGE: English
AVAILABILITY: INIST-21337, 354000065485320100
AN 1997-0307382 PASCAL
CP Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
AB The immunosuppressive activity of human seminal plasma may be one factor in the aetiology of **sexually** transmitted disease and could be particularly important for the spread of human immunodeficiency virus (HIV). The advent of virus that can preferentially infect Langerhans cells of the **genital** mucosa underscores the relevance of

seminal plasma effects. Virally infected cells are eradicated by the killing activity of T cells and natural killer (NK) cells and this cytotoxicity is stimulated by IL-12 (previously known as natural killer cell stimulatory factor) and partly inhibited by IL-10 (previously known as cytokine synthesis inhibitory factor). We have examined the effects of human seminal plasma on the production of these key cytokines. Cytokine production was measured in rapidly diluted, fresh, lipopolysaccharide (LPS)-stimulated, whole blood since this provided leukocytes with minimal exposure to prostaglandin. Prostaglandin concentrations and cytokine release were measured by ELISA. Addition of human seminal plasma diluted up to 100 000 times (0.001%) to blood cell cultures led to a marked increase in the IL-10/IL-12 ratio ($P < 0.02$). A dose-dependent increase in the ratio was observed in five separate experiments, from a control value of 1 (no seminal plasma) to a mean value of 80 (1% seminal plasma). This cytokine switch was also seen when seminal plasma was substituted by pure prostaglandin E (**PGE**) and 19-OH **PGE** (the main prostaglandin constituent of human seminal plasma). Lipid-extracted seminal plasma was considerably less active at high dilutions than whole seminal plasma at the same dilution. However, its activity could be restored by the addition of synthetic **PGE** and 19-hydroxy **PGE**. A stimulation of IL-10 and a decrease in IL-12 in host-defence cells of the lower **female** reproductive tract will seriously affect the ability of cytotoxic T cells and NK cells to recognise and destroy virally infected cells. In addition, the stimulation of IL-10 will inhibit the release of the anti-HIV activity from CD8+ve cells. The cytokine switch reported here, activated by semen deposition, would exercise a key inhibitory control over vital immune defences in the lower **genital** tract, with ablation of cell-mediated **responses** and immunosurveillance.

L29 ANSWER 14 OF 36 PASCAL

ACCESSION NUMBER: 1997-0131769 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Self-reported sexual function and sexual arousability in women with epilepsy
AUTHOR: MORRELL M. J.; GULDNER G. T.
CORPORATE SOURCE: Department of Neurology and Neurological Sciences, Stanford Comprehensive Epilepsy Center, Stanford University School of Medicine, Stanford, California, United States
SOURCE: Epilepsia : (Copenhagen), (1996), 37(12), 1204-1210, 39 refs.
ISSN: 0013-9580 CODEN: EPILAK
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-1145, 354000061163140090
AN 1997-0131769 PASCAL

AB Purpose : **Women** with epilepsy are at risk for **sexual** dysfunction but the frequency and types of dysfunction have not been well characterized. Methods : Self-reported **sexual** function was

evaluated in 116 **women** aged 18-65 years with epilepsy and no concomitant medical or psychiatric illness, including 99 with localization-related epilepsy (LRE) and 17 with primary generalized epilepsy (**PGE**). Variables evaluated included seizure frequency, age of seizure onset, and antiepileptic drug (AED) exposure. Standardized inventories assessed **sexual** functioning, **sexual** arousability and anxiety, **sexual** behavior, and depression. Results.- Although **sexual** experience was not reduced, **women** with **PGE** and LRE reported significantly less **sexual** arousability and **women** with LRE reported significantly more **sexual** anxiety. **Women** with LRE experienced significantly more dyspareunia, vaginismus, **arousal** insufficiency, and **sexual** dissatisfaction, whereas **women** with **PGE** experienced anorgasmia and **sexual** dissatisfaction. **Sexual** symptoms were not associated with seizure frequency, AED exposure, **sexual** experience, depression, or prepubertal seizure onset. Conclusions : In contrast to subjects of previous research, the **women** in our study did not have a disorder of **sexual desire**, but more than one third experienced disorders of **sexual arousal**, implying a physiological deficit. Although the etiology for these **arousal** phase dysfunctions has not been defined, such conditions are treatable and warrant referral to a gynecologist versed in the treatment of **sexual** disorders.

L29 ANSWER 15 OF 36 PASCAL

ACCESSION NUMBER: 1996-0374537 PASCAL

COPYRIGHT NOTICE:

TITLE (IN ENGLISH): Progesterone and dexamethasone stimulate proliferation and differentiation of osteoprogenitors and progenitors for adipocytes and macrophages in cell populations derived from adult rat vertebrae

AUTHOR: ISHIDA Y.; TERTINEGG I.; HEERSCHE J. N. M.

CORPORATE SOURCE: Faculty of Dentistry, University of Toronto, Toronto Ontario, Canada

SOURCE: Journal of bone and mineral research, (1996), 11(7), 921-930, 36 refs.
ISSN: 0884-0431 CODEN: JBMREJ

DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-21114, 354000060117470070

AN 1996-0374537 PASCAL

AB We investigated the effects of the **sex** hormone progesterone (Prog) and the synthetic glucocorticoid dexamethasone (Dex) on proliferation and differentiation of progenitor cells of osteogenic, adipocytic, and hemopoietic lineages in cell populations derived from explants of adult **female** rat lumbar vertebrae. The cell populations were obtained by culturing bone explants in plasma clots immersed in .alpha.-minimum essential medium plus 10% fetal calf serum (standard medium) and then subculturing the outgrowth cells in standard medium plus 50 .mu.g/ml of ascorbic acid, 5 mM .beta.-glycerophosphate, and with or without Prog or Dex. On day 6 of culture, these populations were analyzed for cAMP **responses** to parathyroid hormone (PTH),

prostaglandin E.sub.2 (**PGE**.sub.2), and isoproterenol (IPT). Increases in intracellular cAMP were seen in **response** to PTH, PGH.sub.2, and IPT, and culturing in medium containing Prog increased these **responses**. At various time periods between days 4-27 of culture, the cultures were evaluated for the presence of bone nodules, alkaline phosphatase (AP)-positive colonies, adipocytes, monocytes, and macrophages. Prog and Dex increased the number of bone nodules and AP-positive colonies. The effect of Prog on bone nodule formation was smaller than that of Dex. In addition, the effect of Dex on bone nodule formation was evident after 10 days of culture, while the Prog-induced effects became significant at days 16-20 of culture. Both hormones also increased the number of Sudan IV-positive colonies (adipocytes), certain types of .alpha.-naphthyl butyrate esterase (.alpha.-NBE)-positive colonies (monocytes, macrophages, and T-lymphocytes), and ED2-positive colonies (macrophages). Prog-treated cultures contained more colonies of small spindle-shaped .alpha.-NBE-positive cells and fewer colonies of small round .alpha.-NBE-positive cells when compared with Dex-treated cultures. These data indicate that cell populations derived from adult rat lumbar vertebrae contain, among others, osteoprogenitors and progenitors for adipocytes and macrophages that are stimulated to proliferate and differentiate by Prog and Dex. The data also suggest that the effects of Prog and Dex differ qualitatively and quantitatively.

L29 ANSWER 16 OF 36 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.

ACCESSION NUMBER: 1995-0168851 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1995 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Mesenteric vascular responses to vasopressin during development of DOCA-salt hypertension in male and female rats
AUTHOR: STALLONE J. N.
CORPORATE SOURCE: Northeastern Ohio univ. coll. medicine, dep. physiology, Rootstown OH 44272, United States
SOURCE: American journal of physiology. Regulatory, integrative and comparative physiology, (1995), 37(1), R40-R49, 58 refs.
ISSN: 0363-6119 CODEN: AJPRDO
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-670E, 354000059782010070

AN 1995-0168851 PASCAL
CP Copyright .COPYRGT. 1995 INIST-CNRS. All rights reserved.
AB Deoxycorticosterone acetate (DOCA)-salt hypertension develops to a greater extent in male (M) than in **female** (F) rats. To determine the role of the vasculature, reactivity to arginine vasopressin (AVP) and prostanoid output were examined in the isolated perfused mesenteric vasculature of hypertensive (HT) and normotensive-control (NTC) M and F rats after acute (1-wk) and chronic (4-wk) DOCA-salt treatment. Systolic blood pressure was significantly higher in M than in F HT rats (187.+-.3 vs. 151.+-.3 mmHg after 4 wk; P<0.02). After acute treatment, vascular reactivity to AVP (maximal perfusion pressure) in HT was elevated in M (181.+-.18 mmHg; P<0.02) but not in F (135.+-.6 mmHg) compared with NTC (90.+-.6 mmHg, M vs. 119.+-.5 mmHg, F). After chronic treatment, vascular reactivity to AVP in HT was elevated in both **sexes** (P<0.02), although more in F (175.+-.13 mmHg) than in M

(141.+-.11 mmHg). In contrast, vascular **responsiveness** to phenylephrine did not differ significantly between M and F NTC or HT preparations after either acute or chronic treatment. **Sex** differences in basal and AVP-induced 6-ketoprostaglandin (6-keto-PG) F.sub.1.sub..alpha. and **PGE**.sub.2 output by HT and NTC vasculature were reciprocal to **sex** differences in the vasoconstriction **responses** to AVP. After acute treatment, AVP-stimulated 6-keto-PGF.sub.1.sub..alpha. output by HT was elevated slightly in F (33.6.+-.1.7 ng/3 min; P<=0.02) but not in M (49.9.+-.4.3 ng/3 min) compared with NTC (23.5.+-.2.6 ng/3 min, F vs. 34.7.+-.4.9 ng/3 min, M). After chronic treatment, output by HT was enhanced in both **sexes** (P<=0.02), although more in M (109.+-.15.4 ng/3 min) than in F (68.+-.6.6 ng/3 min). These findings suggest that **sex** differences in the relative balance between AVP-induced vasoconstriction and vasodilatory prostanoid release may contribute to male-**female** differences in mesenteric vascular reactivity to AVP in NT and that disturbances in this balance may be **responsible**, at least in part, for the **sex**- and time-dependent changes in reactivity to AVP observed during the development of DOCA-salt hypertension

L29 ANSWER 17 OF 36 TOXLIT
ACCESSION NUMBER: 1998:15340 TOXLIT
DOCUMENT NUMBER: CA-128-030641P
TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women.
AUTHOR: Farker K; Schweer H; Vollandt R; Nassr N; Nagel U; Seyberth HW; Hoffmann A; Oettel M
CORPORATE SOURCE: Institute of Clinical Pharmacology, Friedrich Schiller University, Jena, Jena
SOURCE: Prostaglandins, (1997). Vol. 54, No. 3, pp. 655-664. CODEN: PRGLBA. ISSN. 0090-6980.
PUB. COUNTRY: GERMANY, FEDERAL REPUBLIC OF
DOCUMENT TYPE: Journal; Journal Article
FILE SEGMENT: CA
LANGUAGE: English
OTHER SOURCE: CA 128:30641
ENTRY MONTH: 199804
AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E2 (PGE2), 6-keto-PGF1.alpha., thromboxane B2 (TxB2) and their metabolites **PGE**-M (11.alpha.-hydroxy-9,15-dioxo-2,3,4,5,20-pentanor-19-carboxyprostanoic acid), 2,3-dinor-6-keto-PGF1.alpha., 2,3-dinor-TxB2 and 11-dehydro-TxB2 were detd. by gas chromatog.-triple stage quadrupole mass spectrometry (GC/MS/MS) in 41 young non-pregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women** . Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, PGE2 and TxB2 were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels of estradiol and progesterone were different in young and postmenopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins.

Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

L29 ANSWER 18 OF 36 TOXLIT
ACCESSION NUMBER: 1988:75313 TOXLIT
DOCUMENT NUMBER: CA-109-067612P
TITLE: Effects of female sex hormones and pregnancy on gallbladder prostaglandin synthesis.
AUTHOR: Hoover EL; Jaffe BM; Webb H; England DW
CORPORATE SOURCE: Health Sci. Cent., State Univ. New York, Brooklyn
SOURCE: Arch. Surg. (Chicago), (1988). Vol. 123, No. 6, pp. 705-8.
CODEN: ARSUAX. ISSN. 0004-0010.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: CA
LANGUAGE: English
OTHER SOURCE: CA 109:67612
ENTRY MONTH: 198810
AB To investigate whether **female sex** hormones and pregnancy induced increased gallbladder synthesis of PGI2 and prostaglandin E (**PGE**), an in vitro incubation chamber was used to quantitate the effects of progesterone, estrogen, pregnancy, and pregnancy plus a 2%-cholesterol diet on mucosal and serosal PGI2 and **PGE** prodn. by the rabbit gallbladder. Neither the **female sex** hormones nor pregnancy alone caused an increase in PGI2 or **PGE** synthesis. The gallbladders of cholesterol-fed, pregnant rabbits demonstrated increases only in serosal synthesis of PGI2. This increased prodn. was equiv. to that noted for gallbladders from nonpregnant rabbits fed a high-cholesterol diet. There were no increases in mucosal synthesis of **PGE** or of PGI2. Thus, neither elevated levels of progesterone or estrogen nor pregnancy is directly **responsible** for the increased PGI2 activity in the **female** gallbladder; conversely, this effect seems to be mediated by the increased biliary concns. of cholesterol.

L29 ANSWER 19 OF 36 TOXLIT
ACCESSION NUMBER: 1982:84100 TOXLIT
DOCUMENT NUMBER: CA-097-139356S
TITLE: Prostaglandin-mediated inhibition of lymphokine secretion in normal individuals and patients with progressive systemic sclerosis (scleroderma, PSS).
AUTHOR: Kelly RH; Miller DH; Rodnan GP; Hagmann J
CORPORATE SOURCE: Sch. Med., Univ. Pittsburgh, Pittsburgh
SOURCE: Agents Actions, (1982). Vol. 12, No. 4, pp. 471-7.
CODEN: AGACBH. ISSN. 0065-4299.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: CA
LANGUAGE: English
OTHER SOURCE: CA 97:139356
ENTRY MONTH: 198211
AB The sensitivity of peripheral blood lymphocytes to E-type prostaglandin (**PGE**)-mediated inhibition of lymphokine secretion was examd. in 3 groups of individuals; normal controls, hospitalized patients, and

patients with progressive systemic sclerosis. Leukocytes were stimulated by a polyclonal T-cell activator, phytohemagglutinin, and the release of the lymphokine, leukocyte migration inhibitory factor (LIF), was measured in the presence or absence of exogenous PGE2 (I) [363-24-6] using a direct agarose droplet migration inhibition technique. Leukocytes of scleroderma patients were hyporesponsive to **PGE** (i.e., lymphokine secretion by these cells was not inhibited at concns. of PGE2 of 2.8 .times. 10-8-2.8 .times. 10-5M). In addn., a marked **sex** difference in **PGE responsiveness** existed among normal controls, whereby **females** were hyporesponsive during the latter half of the menstrual cycle. This deficit may facilitate, in part, the development of connective tissue diseases in **women** of child-bearing age. The inability to suppress lymphokine prodn. and arrest persistent immune reactivity, coupled with the known ability of lymphokines to augment fibroblast collagen prodn., offers a reasonable explanation for the accumulation of tissue collagen in scleroderma.

L29 ANSWER 20 OF 36 TOXLIT
ACCESSION NUMBER: 1981:40993 TOXLIT
DOCUMENT NUMBER: CA-095-018389E
TITLE: Effect of adrenoceptor blockers and MAOI on postcoital utero-oviductal contractility, sperm transport, and sperm attachment to eggs in rabbits.
AUTHOR: Marsafy YM; Hafez ES E
CORPORATE SOURCE: Sch. Med., Wayne State Univ., Detroit
SOURCE: Arch. Androl, (1981). Vol. 6, No. 4, pp. 307-16. CODEN: ARANDR.
FILE SEGMENT: CA
LANGUAGE: English
OTHER SOURCE: CA 95:18389
ENTRY MONTH: 198108

AB Adult **female** rabbits were injected with phenoxybenzamine (alpha-adrenoceptor blocker), propranolol (beta-adrenoceptor blocker), and isocarboxazid (monoamine oxidase inhibitor, MAOI). (59-96-1 Phenoxybenzamine)(525-66-6 Propranolol)(59-63-2 Isocarboxazid) Physiol. saline was injected in the controls. Treatment started 1 day before mating and continued until autopsy. At 3, 12, and 24 h postcoitum (PC), utero-oviductal contractions were recorded in vivo using micro-balloon-ended, fluid-filled catheters as pressure receptors. At 24 h PC, all groups were autopsied and flushings of the contralateral oviducts were evaluated for both sperm count and no. of sperm attached and/or penetrating the eggs. Phenoxybenzamine induced redn. in the amplitude and frequency of both active contractions and resting pressure fluctuations. This suppressive effect was more remarkable at 12 and 24 h PC than at 3 h PC. Propranolol induced incoordination and instability in the uterooviductal resting pressure and increased the amplitude of isthmic contraction, and in frequency of uterine and ampullary contractions. Such excitatory effects were more pronounced at 3 h PC than at 12 and 24 h PC. More effective alpha-adrenergic blockade seemed to coincide with postovulatory progesterone dominance, increase in concn., and **response** to **genital** tract **PGE**. More effective beta-adrenoceptor blockade coincided with preovulatory estrogen dominance and increased concn. and **response** to **genital** tissue PGF. At 24 h PC, sperm count was reduced in the oviduct and fewer nos. of sperm were attached to the eggs in phenoxybenzamine- and

propranolol-treated **females**. Phenoxybenzamine-induced suppression of utero-oviductal contractions facilitated oviductal sperm ascent. Propranolol caused irregular sperm transport with rapid loss in the peritoneal cavity. At 24 h PC, oviductal sperm count increased and an excessive no. of sperm were attached to the eggs in isocarboxazid-treated rabbits. The pharmacol. of sperm transport is discussed in relation to infertility and contraception.

=>

=> d 21-30 ibib abs

L29 ANSWER 21 OF 36 TOXLIT
ACCESSION NUMBER: 1981:31132 TOXLIT
DOCUMENT NUMBER: CA-094-167790K
TITLE: Effects of cannabinoids and female exposure on the pituitary-testicular axis in mice: possible involvement of prostaglandins.
AUTHOR: Dalterio S; Bartke A; Harper MJ K; Huffman R; Sweeney C
CORPORATE SOURCE: Health Sci. Cent., Univ. Texas, San Antonio
SOURCE: Biol. Reprod, (1981). Vol. 24, No. 2, pp. 315-22.
CODEN: BIREBV.
FILE SEGMENT: CA
LANGUAGE: English
OTHER SOURCE: CA 94:167790
ENTRY MONTH: 198106

AB In immature (30-35-day-old) mice, a single dose of DELTA9-THC(I), the main psychoactive constituent of marihuana, decreased plasma testosterone (T), LH, and FSH levels, but the same dose of a nonpsychoactive component, cannabinol (CBN), had not effect. (1972-08-3 .DELTA.9-Tetrahydrocannabinol)(58-22-0 Testosterone)(9002-67-9 LH)(9002-68-0 FSH)(521-35-7 Cannabinol) Chronic exposure to THC, CBN, or cannabidiol (CBD), beginning at 30 days of age through adulthood, influenced the endocrine **responses** to a **sexually** receptive **female**. (13956-29-1 Cannabidiol) Thus, wts. of testes and seminal vesicles were reduced in males from all cannabinoid-treated groups on the day after exposure to a **female**, compared with treated males housed in all-male groups. Plasma FSH concns. were elevated in CBN-exposed mice, regardless of social experience, while plasma T levels were increased after an encounter with a **female** in all but THC-treated males. Plasma LH levels and testicular **responsiveness** to gonadotropins in vitro were reduced in THC- and CBN-treated mice exposed to a **female**. In contrast, in THC- or CBN-treated males maintained in all-male groups, T prodn. in vitro was significantly elevated. Alterations in prostaglandin (PG) concns. may mediate these effects of cannabinoids and **sexual** encounter since prodn. of PG in vitro by testis and pituitary was reduced by exposing cannabinoid-treated males to **female**-related stimuli. In contrast, **sexual** encounter increased PGF, but had no effect on **PGE** prodn. by pituitary or testes obtained from oil-treated controls. Both psychoactive and nonpsychoactive constituents of marihuana are capable of altering the function of the pituitary-gonadal axis and of influencing the endocrine **responsivity** to **female**-related exteroceptive cues in male mice.

L29 ANSWER 22 OF 36 Elsevier BIOBASE COPYRIGHT 2001 Elsevier Science B.V.
ACCESSION NUMBER: 1997256480 Elsevier BIOBASE
TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women
AUTHOR: Farker K.; Schweer H.; Vollandt R.; Nassr N.; Nagel U.; Seyberth H.W.; Hoffmann A.; Oettel M.
CORPORATE SOURCE: Dr. K. Farker, Institute of Clinical Pharmacology, Friedrich-Schiller-University Jena, D-07740 Jena, Germany.
SOURCE: Prostaglandins, (1997), 54/3 (655-664), 18 reference(s)
CODEN: PRGLBA ISSN: 0090-6980
PUBLISHER ITEM IDENT.: S0090698097001317
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E.sub.2 (**PGE**.sub.2), 6-keto-PGF.sub.1.alpha., thromboxane B.sub.2 (TXB.sub.2) and their metabolites **PGE**-M (11.alpha.- hydroxy-9,15-dioxo-2,3,4,5,20-pentanor-19-carboxyprostanoic acid), 2,3- dinor-6-keto-PGF.sub.1.alpha., 2,3-dinor-TXB.sub.2 and 11-dehydro-TxB.sub.2 were determined by gas chromatography-triple stage quadrupole mass spectrometry (GC/MS/MS) in 41 young non pregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women**. Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, **PGE**.sub.2 and TxB.sub.2 were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels of estradiol and progesterone were different in young and postmenopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins. Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

L29 ANSWER 23 OF 36 Elsevier BIOBASE COPYRIGHT 2001 Elsevier Science B.V.
ACCESSION NUMBER: 1997133358 Elsevier BIOBASE
TITLE: Differential ventral septal vasopressin release is associated with sexual dimorphism in PGE.sub.2 fever
AUTHOR: Chen X.; Landgraf R.; Pittman Q.J.
CORPORATE SOURCE: X. Chen, Neuroscience Research Group, Dept. of Physiology and Biophysics, University of Calgary, 3330 Hospital Dr. NW, Calgary, Alta. T2N 4N1, Canada.
SOURCE: American Journal of Physiology - Regulatory Integrative and Comparative Physiology, (1997), 272/5 41-5 (R1664-R1669), 30 reference(s)
CODEN: AJPRDO ISSN: 0363-6119

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The vasopressinergic innervation of the ventral septal area (VSA) has been shown to be implicated in antipyresis. Because this system is less well developed in **female** rats, we hypothesized that **female** rats would display exaggerated febrile **responses**. We therefore examined the temperature **responses** of conscious and urethan-anesthetized rats of both **sexes** to centrally administered prostaglandin E.sub.2 (**PGE**.sub.2) and correlated these **responses** with the release and action of endogenous arginine vasopressin (AVP) in the VSA. Both conscious .cents.25 ng/5 .mu.l **PGE**.sub.2 intracerebroventricularly (icv)! and anesthetized (VSA microdialyzed, 50 ng/5 .mu.l **PGE**.sub.2 icv) **female** rats had higher fevers than did males. Infusion of an AVP V(1a) receptor antagonist .cents.1 nmol! d(CH.sub.2).sub.5Tyr(Me)!AVP! plus **PGE**.sub.2 gave rise to higher fevers in males but not in **females**. Measurements of AVP in microdialysates of the VSA showed that the release of endogenous AVP was increased in **response** to **PGE**.sub.2 in males only. Baseline AVP release in both **sexes** was similar. The results suggest that there is a **sex**-related difference in **PGE**.sub.2 fever, which may be accounted for by the differential AVP release in the VSA.

L29 ANSWER 24 OF 36 Elsevier BIOBASE COPYRIGHT 2001 Elsevier Science B.V.
ACCESSION NUMBER: 1996190112 Elsevier BIOBASE
TITLE: Self-reported sexual function and sexual arousability in women with epilepsy
AUTHOR: Morrell M.J.; Guldner G.T.
CORPORATE SOURCE: Dr. M.J. Morrell, Dept. of Neurology/Neurological Sci., Stanford University Medical Center, Stanford, CA 94307, United States.
SOURCE: Epilepsia, (1996), 37/12 (1204-1210)
CODEN: EPILAK ISSN: 0013-9580
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Purpose: **Women** with epilepsy are at risk for **sexual** dysfunction but the frequency and types of dysfunction have not been well characterized. Methods: Self-reported **sexual** function was evaluated in 116 **women** aged 18-65 years with epilepsy and no concomitant medical or psychiatric illness, including 99 with localization-related epilepsy (LRE) and 17 with primary generalized epilepsy (**PGE**). Variables evaluated included seizure frequency, age of seizure onset, and antiepileptic drug (AED) exposure. Standardized inventories assessed **sexual** functioning, **sexual** arousability and anxiety, **sexual** behavior, and depression. Results: Although **sexual** experience was not reduced, **women** with **PGE** and LRE reported significantly less **sexual** arousability and **women** with LRE reported significantly more **sexual** anxiety. **Women** with LRE experienced significantly more dyspareunia, vaginismus, **arousal** insufficiency, and **sexual** dissatisfaction, whereas **women** with **PGE** experienced anorgasmia and **sexual** dissatisfaction. **Sexual** symptoms were not

associated with seizure frequency, AED exposure, **sexual** experience, depression, or prepubertal seizure onset. Conclusions: In contrast to subjects of previous research, the **women** in our study did not have a disorder of **sexual desire**, but more than one third experienced disorders of **sexual arousal**, implying a physiological deficit. Although the etiology for these **arousal** phase dysfunctions has not been defined, such conditions are treatable and warrant referral to a gynecologist versed in the treatment of **sexual** disorders.

L29 ANSWER 25 OF 36 LIFESCI COPYRIGHT 2001 CSA
ACCESSION NUMBER: 1998:33681 LIFESCI
TITLE: Enhancement by sex hormones of the osteoregulatory effects of mechanical loading and prostaglandins in explants of rat ulnae
AUTHOR: Cheng, Ming Zhao; Zaman, G.; Rawlinson, S.C.F.; Pitsillides, A.A.; Suswillo, R.F.L.; Lanyon, L.E.*
CORPORATE SOURCE: Royal Veterinary College, University of London, Royal College Street, London NW1 0TU, UK
SOURCE: J. BONE MINER. RES., (19970900) vol. 12, no. 9, pp. 1424-1430.
ISSN: 0884-0431.
DOCUMENT TYPE: Journal
FILE SEGMENT: T
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Explants of ulnae from 5-week-old male and **female** rats were cleaned of marrow and soft tissue and, in the presence and absence of 10 super(-8) M 17 beta -estradiol (E2) or 5 alpha -dihydrotestosterone (DHT), mechanically loaded or treated with exogenous prostanoids previously shown to be produced during loading. Over an 18-h period, mechanical loading (peak strain 1300 mu epsilon , 1 Hz, 8 minutes, maximum strain rate 25,000 mu epsilon /s), prostaglandin E sub(2) (**PGE** sub(2)) and prostacyclin (PGI sub(2)) (10 super(-6) M), each separately produced quantitatively similar increases in cell proliferation and matrix production in bones from males and **females**, as indicated by incorporation of [super(3)H]thymidine into DNA and [super(3)H]proline into collagen. E2 and DHT both increased [super(3)H]thymidine and [super(3)H]proline incorporations, E2 producing greater increases in **females** than in males. Indomethacin abrogated the effects of loading, but had no effects on those of **sex** hormones. Loading, or prostanoids, together with **sex** hormones, produced **responses** generally equal to or greater than the addition of the individual influences acting independently. In **females** there was a synergistic **response** in [super(3)H]thymidine incorporation between loading and E2, which was quantitatively similar to the interaction between E2 and **PGE** sub(2) or PGI sub(2). The interaction between loading and E2 for [super(3)H]proline incorporation was not mimicked by these prostanoids. In males the synergism in [super(3)H]proline incorporation seen between loading and DHT was mimicked by that between PGI sub(2) and DHT. We conclude that loading stimulates increased bone cell proliferation and matrix production in situ through a prostanoid-dependent mechanism. This **response** is equal in size in males and **females**. Estrogen and testosterone increase proliferation and matrix production through a mechanism independent of prostanoid production. The interactions between loading and hormones are

reproduced in some but not all cases by E2 and prostaglandins. E2 with loading and prostaglandins has greater effects in **female** bones, while DHT with loading and prostaglandins has greater effects in males.

L29 ANSWER 26 OF 36 LIFESCI COPYRIGHT 2001 CSA
ACCESSION NUMBER: 97:14091 LIFESCI
TITLE: Progesterone and dexamethasone stimulate proliferation and differentiation of osteoprogenitors and progenitors for adipocytes and macrophages in cell populations derived from adult rat vertebrae
AUTHOR: Ishida, Y.; Tertinegg, I.; Heersche, J.N.M.
CORPORATE SOURCE: Faculty of Dentistry, University of Toronto, Room 400, 124 Edward Street, Toronto, Ontario M5G 1G6, Canada
SOURCE: J. BONE MINER. RES., (1996) vol. 11, no. 7, pp. 921-930. ISSN: 0884-0431.
DOCUMENT TYPE: Journal
FILE SEGMENT: T
LANGUAGE: English
SUMMARY LANGUAGE: English

AB We investigated the effects of the **sex** hormone progesterone (Prog) and the synthetic glucocorticoid dexamethasone (Dex) on proliferation and differentiation of progenitor cells of osteogenic, adipocytic, and hemopoietic lineages in cell populations derived from explants of adult **female** rat lumbar vertebrae. The cell populations were obtained by culturing bone explants in plasma clots immersed in alpha -minimum essential medium plus 10% fetal calf serum (standard medium) and then subculturing the outgrowth cells in standard medium plus 50 mu g/ml of ascorbic acid, 5 mM beta -glycerophosphate, and with or without Prog or Dex. On day 6 of culture, these populations were analyzed for cAMP **responses** to parathyroid hormone (PTH), prostaglandin E sub(2) (**PGE** sub(2)), and isoproterenol (IPT). Increases in intracellular cAMP were seen in **response** to PTH, **PGE** sub(2), and IPT, and culturing in medium containing Prog increased these **responses**. At various time periods between days 4-27 of culture, the cultures were evaluated for the presence of bone nodules, alkaline phosphatase (AP)-positive colonies, adipocytes, monocytes, and macrophages. Prog and Dex increased the number of bone nodules and AP-positive colonies. The effect of Prog on bone nodule formation was smaller than that of Dex. In addition, the effect of Dex on bone nodule formation was evident after 10 days of culture, while the Prog-induced effects became significant at days 16-20 of culture. Both hormones also increased the number of Sudan IV-positive colonies (adipocytes), certain types of alpha -naphthyl butyrate esterase (alpha -NBE)-positive colonies (monocytes, macrophages, and T-lymphocytes), and ED2-positive colonies (macrophages). Prog-treated cultures contained more colonies of small spindle-shaped alpha -NBE-positive cells and fewer colonies of small round alpha -NBE-positive cells when compared with Dex-treated cultures. These data indicate that cell populations derived from adult rat lumbar vertebrae contain, among others, osteoprogenitors and progenitors for adipocytes and macrophages that are stimulated to proliferate and differentiate by Prog and Dex. The data also suggest that the effects of Prog and Dex differ qualitatively and quantitatively.

L29 ANSWER 27 OF 36 LIFESCI COPYRIGHT 2001 CSA
ACCESSION NUMBER: 81:28933 LIFESCI
TITLE: Effect of Specific Estrogens on Prostaglandin Synthesis in Aorta and Thrombocytes of Female Pigeons.
AUTHOR: Subbiah, M.T.R.; Deitemeyer, D.; Yunker, R.; Gallon, L.
CORPORATE SOURCE: Dept. Med. Pathol., Lipid Res. Ctr., Univ. Cincinnati Med.

Ctr., Cincinnati, OH 45267, USA
SOURCE: PROC. SOC. EXP. BIOL. MED., (1981) vol. 166, no. 2, pp. 300-304.
DOCUMENT TYPE: Journal
FILE SEGMENT: X
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The effect of two major natural estrogens (estrone and 17 beta -estradiol) on prostaglandin biosynthesis from (super(14)C)arachidonic acid in thrombocytes and aorta of **female** pigeons was compared with that of a male **sex** hormone (testosterone). In the aorta, 17 beta -estradiol stimulated the synthesis of 6-keto, PGF sub(1 alpha) and PGF sub(2 alpha) but markedly reduced the synthesis of **PGE** sub(2). Estrone on the other hand stimulated the synthesis of **PGE** sub(2). Testosterone stimulated the synthesis of all prostaglandins in the aorta. In the thrombocytes, 17 beta -estradiol decreased aggregatory **response** to arachidonic acid and synthesis of thromboxane B sub(2). Estrone on the other hand increased aggregatory **response** to arachidonic acid. Testosterone decreased the synthesis of thromboxane B sub(2).

L29 ANSWER 28 OF 36 CANCERLIT
ACCESSION NUMBER: 97302992 CANCERLIT
DOCUMENT NUMBER: 97302992
TITLE: A cytokine switch induced by human seminal plasma: an immune modulation with implications for sexually transmitted disease.
AUTHOR: Kelly R W; Carr G G; Critchley H O
CORPORATE SOURCE: Medical Research Council Reproductive Biology Unit, University of Edinburgh Centre for Reproductive Biology, UK
SOURCE: HUMAN REPRODUCTION, (1997). Vol. 12, No. 4, pp. 677-81. Journal code: HRP. ISSN: 0268-1161.
DOCUMENT TYPE: (CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 97302992
ENTRY MONTH: 199708
AB The immunosuppressive activity of human seminal plasma may be one factor in the aetiology of **sexually** transmitted disease and could be particularly important for the spread of human immunodeficiency virus (HIV). The advent of virus that can preferentially infect Langerhans cells of the **genital** mucosa underscores the relevance of seminal plasma effects. Virally infected cells are eradicated by the killing activity of T cells and natural killer (NK) cells and this cytotoxicity is stimulated by IL-12 (previously known as natural killer cell stimulatory factor) and partly inhibited by IL-10 (previously known as cytokine synthesis inhibitory factor). We have examined the effects of human seminal plasma on the production of these key cytokines. Cytokine production was measured in rapidly diluted, fresh, lipopolysaccharide (LPS)-stimulated, whole blood since this provided leukocytes with minimal exposure to prostaglandin. Prostaglandin concentrations and cytokine release were measured by ELISA. Addition of human seminal plasma diluted

up to 100,000 times (0.001%) to blood cell cultures led to a marked increase in the IL-10/IL-12 ratio (P <0.02). A dose-dependent increase in the ratio was observed in five separate experiments, from a control value of 1 (no seminal plasma) to a mean value of 80 (1% seminal plasma). This cytokine switch was also seen when seminal plasma was substituted by pure prostaglandin E (**PGE**) and 19-OH **PGE** (the main prostaglandin constituent of human seminal plasma). Lipid-extracted seminal plasma was considerably less active at high dilutions than whole seminal plasma at the same dilution. However, its activity could be restored by the addition of synthetic **PGE** and 19-hydroxy **PGE**. A stimulation of IL-10 and a decrease in IL-12 in host-defence cells of the lower **female** reproductive tract will seriously affect the ability of cytotoxic T cells and NK cells to recognise and destroy virally infected cells. In addition, the stimulation of IL-10 will inhibit the release of the anti-HIV activity from CD8+ve cells. The cytokine switch reported here, activated by semen deposition, would exercise a key inhibitory control over vital immune defences in the lower **genital** tract, with ablation of cell-mediated **responses** and immunosurveillance.

L29 ANSWER 29 OF 36 CANCERLIT
ACCESSION NUMBER: 83097004 CANCERLIT
DOCUMENT NUMBER: 83097004
TITLE: Prostaglandin-mediated inhibition of lymphokine secretion in normal individuals and patients with progressive systemic sclerosis (scleroderma, PSS).
AUTHOR: Kelly R H; Miller D H; Rodnan G P; Hagmann J
CONTRACT NUMBER: FR-00056
SOURCE: AGENTS AND ACTIONS, (1982). Vol. 12, No. 4, pp. 471-7. Journal code: 2XZ. ISSN: 0065-4299.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 83097004
ENTRY MONTH: 198303

AB The sensitivity of peripheral blood lymphocytes to E-type prostaglandin-mediated inhibition of lymphokine secretion was examined in 3 groups of individuals; normal controls, hospitalized patients, and patients with progressive systemic sclerosis (PSS, scleroderma). Leukocytes were stimulated by a polyclonal T-cell activator, phytohemagglutinin, and the release of the lymphokine, leukocyte migration inhibitory factor (LIF), was measured in the presence or absence of exogenous PGE2 using a direct agarose droplet migration inhibition technique. Leukocytes of scleroderma patients were found to be hyporesponsive to E-type prostaglandin (i.e., lymphokine secretion by these cells was not inhibited at concentrations of PGE2 between 2.8 X 10(-8) and 2.8 X 10(-5) M). In addition, a marked **sex** difference in **PGE responsiveness** was found to exist among normal controls, whereby **females** were hyporesponsive during the latter half of the menstrual cycle. It is possible that this deficit may facilitate, in part, the development of connective tissue diseases in **women** of childbearing age. The inability to suppress lymphokine production and arrest persistent immune reactivity, coupled with the known ability of lymphokines to augment fibroblast collagen production, offers a a reasonable explanation for the accumulation of tissue collagen in scleroderma.

L29 ANSWER 30 OF 36 DRUGU COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1998-15025 DRUGU T E
TITLE: Treatment of endometrial adenomatosis with tamoxifen in patients of reproductive age.
AUTHOR: Smetnik V P; Chernukha G E; Kushlinskii N E
CORPORATE SOURCE: Russian-Acad.Med.Sci.
LOCATION: Moscow, Russia
SOURCE: Byull.Eksp.Biol.Med. (125, No. 1, 93-97, 1998) 2 Tab. 18 Ref.
CODEN: BEBMAE ISSN: 0365-9615
AVAIL. OF DOC.: Scientific Center for Obstetrics, Gynecology and Perinatology, Russian Academy of Medical Sciences, Moscow, Russia.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AN 1998-15025 DRUGU T E
AB Courses of tamoxifen (TA) were effective against recurrent endometrial hyperplasia and adenomatosis in most of 51 **women** of reproductive age who had previously failed to **respond** to hormone therapy. Initial studies showed that most patients had reductions in serum **sex** hormone binding globulin (SHBG), resulting in increased free blood estrogen levels; endometrial **PGE** levels were also elevated. In patients with complete morphological **responses** to TA, these changes were reversed and estrogen-17-beta (ES) receptors disappeared from the endometrium. However, in patients with partial or no **responses**, changes were smaller or absent, and in some cases total ES increased. While all **women** were initially infertile, pregnancy was established in 2 following TA treatment. TA produced no side-effects.
ABEX Methods 51 Women (mean age 30.9 yr) with recurrent endometrial hyperplasia and adenomatosis received TA (30 mg/day) for 3 mth, extended to 6 mth when incomplete responses were obtained. Controls were 11 healthy women (mean age 28.6 yr). Results Baseline studies showed that that 76.6% of patients were overweight, 70.5% were hirsute, all had chronic anovulation, and 29.4% had cystic ovaries. Gonadotrophic hormones were essentially normal, though the LH/FSH ratio was elevated in 29.4%. Total serum ES was normal but, because of reductions in SHBG in 86.3% of cases, free ES levels were elevated nearly 2-fold. ES receptors were present in higher concentrations than in controls. Thus, patients were considered to have hyperestrogenemia resulting in hyperplasia. TA was followed by amenorrhea in 47.1% and regular or rare menstruation in 52.0%. Among the former and latter groups, TA eliminated adenomatosis in 100% and 85.2% and eliminated all signs of hyperplasia in 62.5% and 41%; 1 patient in each group became pregnant. After extension of treatment to 6 mth in patients with partial or no responses, only 3 remained with focal hyperplasia and 1 with focal adenomatosis requiring surgery. SHBG levels and free ES levels normalized in patients with good morphological responses but not in non-responders. (VH)

=> fil stng
COST IN U.S. DOLLARS SINCE FILE TOTAL

	ENTRY	SESSION
FULL ESTIMATED COST	133.51	233.43

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'STNGUIDE' ENTERED AT 12:49:45 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May 11, 2001 (20010511/UP).

=> FIL F2, F6-F9, F11-F22

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.00	233.43

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'SCISEARCH' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 Institute for Scientific Information (ISI) (R)

FILE 'PASCAL' ENTERED AT 12:50:16 ON 14 MAY 2001
Any reproduction or dissemination in part or in full,
by means of any process and on any support whatsoever
is prohibited without the prior written agreement of INIST-CNRS.
COPYRIGHT (C) 2001 INIST-CNRS. All rights reserved.

FILE 'BIOTECHNO' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'TOXLIT' ENTERED AT 12:50:16 ON 14 MAY 2001

FILE 'ESBIOBASE' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'LIFESCI' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 Cambridge Scientific Abstracts (CSA)

FILE 'PROMT' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 Gale Group. All rights reserved.

FILE 'CANCERLIT' ENTERED AT 12:50:16 ON 14 MAY 2001

FILE 'DRUGU' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

FILE 'TOXLINE' ENTERED AT 12:50:16 ON 14 MAY 2001

FILE 'CABA' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 CAB INTERNATIONAL (CABI)

FILE 'ADISINSIGHT' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

```
FILE 'DRUGUPDATES' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 IMSWORLD Publications Ltd

FILE 'WPIDS' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

FILE 'WPINDEX' TEMPORARILY UNAVAILABLE

FILE 'ADISALERTS' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

FILE 'DDFU' TEMPORARILY UNAVAILABLE
If this message appears repeatedly, please notify the Help Desk.
Enter "HELP STN" for information on contacting the nearest STN Help
Desk by telephone or via SEND in the STNMAIL file.

=> d 31-36 ibib abs
NO VALID FORMATS ENTERED FOR FILE 'ADISINSIGHT'
NO VALID FORMATS ENTERED FOR FILE 'DRUGUPDATES'
In a multifile environment, each file must have at least one valid
format requested.  Refer to file specific help messages or the
STNGUIDE file for information on formats available in individual
files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> d his

     (FILE 'HOME' ENTERED AT 11:31:54 ON 14 MAY 2001)

     FILE 'REGISTRY' ENTERED AT 11:33:36 ON 14 MAY 2001
L1             1 S MISOPROSTOL/CN

     FILE 'STNGUIDE' ENTERED AT 11:34:08 ON 14 MAY 2001

     FILE 'HCAPLUS' ENTERED AT 11:35:06 ON 14 MAY 2001
L2           882 S MISOPROSTOL OR L1
L3           145 S (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENI
L4         27636 S SCAN
L5           595 S ((SEX? OR GENITAL) (A) (HYPOACTIV? OR DESIR? OR SATISFACT?
OR
L6         12767 S ((SEX? OR GENITAL) (S) (HYPOACTIV? OR DESIR? OR SATISFACT?
OR
L7          1933 S (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENI
L8             2 S L2 AND L7

     FILE 'STNGUIDE' ENTERED AT 11:42:34 ON 14 MAY 2001

     FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS, USPATFULL' ENTERED AT 11:52:55 ON
     14 MAY 2001
                E NEAL, GARY/AU
                E NEAL GARY/AU
                E E18
L9            10 S E18

     FILE 'MEDLINE, EMBASE, BIOSIS, USPATFULL' ENTERED AT 11:57:31 ON 14 MAY
     2001
L10        43446 S (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENI
```

```
L11         6378 S L1 OR MISOPROSTOL
L12            1 S L10 AND L11

     FILE 'STNGUIDE' ENTERED AT 12:00:21 ON 14 MAY 2001

     INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS,
     BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT,
     CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE,
     DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, ...' ENTERED AT 12:02:52 ON 14
MAY
     2001

     FILE 'MEDLINE, EMBASE, BIOSIS, USPATFULL' ENTERED AT 12:03:09 ON 14 MAY
     2001
L13        23010 S L1 OR MISOPROSTOL OR (PROSTAGLADIN E) OR PGE
L14           55 S L13 AND L10
L15           41 DUP REM L14 (14 DUPLICATES REMOVED)
L16           37 S L13 (S) L10
L17           23 DUP REM L16 (14 DUPLICATES REMOVED)
L18           21 S L17 1999
L19           19 S L17 1998

     FILE 'STNGUIDE' ENTERED AT 12:16:42 ON 14 MAY 2001

     INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS,
     BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT,
     CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE,
     DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, ...' ENTERED AT 12:20:12 ON 14
MAY
     2001
               SEA L1 OR MISOPROSTOL OR (PROSTAGLADIN E) OR PGE
              ---------
            1241*  FILE ADISALERTS
              39   FILE ADISINSIGHT
              81   FILE AGRICOLA
              24   FILE ANABSTR
             104*  FILE AQUASCI
             172   FILE BIOBUSINESS
               2*  FILE BIOCOMMERCE
            9147   FILE BIOSIS
              15   FILE BIOTECHABS
              15   FILE BIOTECHDS
            4490   FILE BIOTECHNO
             439*  FILE CABA
             808   FILE CANCERLIT
            5060*  FILE CAPLUS
              35*  FILE CEABA-VTB
               1   FILE CEN
              76   FILE CIN
             361*  FILE CONFSCI
               0*  FILE CROPB
               2*  FILE CROPU
             120*  FILE DDFB
            2488*  FILE DDFU
              61*  FILE DGENE
             120*  FILE DRUGB
             121   FILE DRUGLAUNCH
             125   FILE DRUGMONOG2
              19   FILE DRUGNL
            2874*  FILE DRUGU
```

```
   14   FILE DRUGUPDATES
  171*  FILE EMBAL
 5933   FILE EMBASE
 3570*  FILE ESBIOBASE
    0*  FILE FOMAD
    0*  FILE FOREGE
   11*  FILE FROSTI
   14   FILE FSTA
   25*  FILE GENBANK
   26*  FILE HEALSAFE
  259*  FILE IFIPAT
  282   FILE JICST-EPLUS
    3*  FILE KOSMET
 3307*  FILE LIFESCI
    1*  FILE MEDICONF
 4475   FILE MEDLINE
   45   FILE NIOSHTIC
   84*  FILE NTIS
   23*  FILE OCEAN
 5188*  FILE PASCAL
   30   FILE PHAR
    0*  FILE PHIC
  476*  FILE PHIN
 1371   FILE PROMT
 8868*  FILE SCISEARCH
    6   FILE SYNTHLINE
 1693   FILE TOXLINE
 2279   FILE TOXLIT
 3435*  FILE USPATFULL
  375   FILE WPIDS
  375   FILE WPINDEX
L20          QUE L1 OR MISOPROSTOL OR (PROSTAGLADIN E) OR PGE
             ---------
             SEA (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GE
             ---------
  283   FILE ADISALERTS
   69   FILE ADISINSIGHT
  832   FILE AGRICOLA
    2   FILE ANABSTR
  734   FILE AQUASCI
  303   FILE BIOBUSINESS
    2   FILE BIOCOMMERCE
15038   FILE BIOSIS
   12   FILE BIOTECHABS
   12   FILE BIOTECHDS
 1825   FILE BIOTECHNO
 4136   FILE CABA
 2254   FILE CANCERLIT
 1933   FILE CAPLUS
    2   FILE CEABA-VTB
   11   FILE CEN
    9   FILE CIN
   23   FILE CONFSCI
  117   FILE CROPB
  374   FILE CROPU
   96   FILE DDFB
  421   FILE DDFU
  100   FILE DGENE
   96   FILE DRUGB
```

```
     8   FILE DRUGNL
  1532   FILE DRUGU
    14   FILE DRUGUPDATES
   152   FILE EMBAL
 13934   FILE EMBASE
  3509   FILE ESBIOBASE
     2   FILE FOMAD
    21   FILE FROSTI
    61   FILE FSTA
     1   FILE GENBANK
   231   FILE HEALSAFE
    57   FILE IFIPAT
   748   FILE JICST-EPLUS
    15   FILE KOSMET
  4182   FILE LIFESCI
     2   FILE MEDICONF
 13531   FILE MEDLINE
   561   FILE NIOSHTIC
   347   FILE NTIS
   185   FILE OCEAN
  4100   FILE PASCAL
     9   FILE PHAR
     1   FILE PHIC
    64   FILE PHIN
  1233   FILE PROMT
  7404   FILE SCISEARCH
  3855   FILE TOXLINE
  3780   FILE TOXLIT
   943   FILE USPATFULL
    91   FILE WPIDS
    91   FILE WPINDEX
L21          QUE (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GE
             ---------
             SEA L20 AND L21
             ---------
             1*  FILE ADISALERTS
             1   FILE ADISINSIGHT
             0*  FILE AQUASCI
             0*  FILE BIOCOMMERCE
            19   FILE BIOSIS
             8   FILE BIOTECHNO
             2*  FILE CABA
             4   FILE CANCERLIT
             7*  FILE CAPLUS
             0*  FILE CEABA-VTB
             0*  FILE CONFSCI
             0*  FILE CROPB
             0*  FILE CROPU
             0*  FILE DDFB
             1*  FILE DDFU
             0*  FILE DGENE
             0*  FILE DRUGB
             3*  FILE DRUGU
             1   FILE DRUGUPDATES
             0*  FILE EMBAL
            11   FILE EMBASE
             8*  FILE ESBIOBASE
             0*  FILE FOMAD
             0*  FILE FOREGE
```

```
                0*  FILE FROSTI
                0*  FILE GENBANK
                0*  FILE HEALSAFE
                0*  FILE IFIPAT
                0*  FILE KOSMET
                6*  FILE LIFESCI
                0*  FILE MEDICONF
               10   FILE MEDLINE
                0*  FILE NTIS
                0*  FILE OCEAN
               10*  FILE PASCAL
                0*  FILE PHIC
                0*  FILE PHIN
                5   FILE PROMT
               18*  FILE SCISEARCH
                2   FILE TOXLINE
                8   FILE TOXLIT
               15*  FILE USPATFULL
                1   FILE WPIDS
                1   FILE WPINDEX
L22              QUE L20 AND L21
                 ---------

     FILE 'SCISEARCH, PASCAL, BIOTECHNO, TOXLIT, ESBIOBASE, LIFESCI, PROMT,
     CANCERLIT, DRUGU, TOXLINE, CABA, ADISINSIGHT, DRUGUPDATES, WPIDS,
     ADISALERTS' ENTERED AT 12:27:36 ON 14 MAY 2001
L23           605 S F22
L24            79 S L22
L25            69 S L20 (S) L21
L26        222051 S CELLULOSE
L27             0 S L26 (S) L25
L28             0 S L26 AND L25
L29            36 S L25
```

```
     FILE 'STNGUIDE' ENTERED AT 12:49:45 ON 14 MAY 2001

     FILE 'SCISEARCH, PASCAL, BIOTECHNO, TOXLIT, ESBIOBASE, LIFESCI, PROMT,
     CANCERLIT, DRUGU, TOXLINE, CABA, ADISINSIGHT, DRUGUPDATES, WPIDS,
     ADISALERTS' ENTERED AT 12:50:16 ON 14 MAY 2001

=> d l29 31-36 ibib abs
NO VALID FORMATS ENTERED FOR FILE 'ADISINSIGHT'
NO VALID FORMATS ENTERED FOR FILE 'DRUGUPDATES'
In a multifile environment, each file must have at least one valid
format requested.  Refer to file specific help messages or the
STNGUIDE file for information on formats available in individual
files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> d l29 31-36

L29   ANSWER 31 OF 36  DRUGU  COPYRIGHT 2001 DERWENT INFORMATION LTD
AN    1998-10539  DRUGU   T E
TI    Antigestagens: Possible gynecological and obstetric uses.
AU    Ortmann O; Emons G; Schulz K D
CS    Univ.Philipps-Marburg; Univ.Lubeck
LO    Lubeck; Marburg, Ger.
SO    Med.Welt (48, No. 12, 525-29, 1997) 1 Fig. 39 Ref.
      CODEN: MEWEAC       ISSN:  0025-8512
```

AV Medizinische Universitaet zu Luebeck, Ratzeburgerallee 160, D-23562 Luebeck, Germany.
LA German
DT Journal
FA AB; LA; CT
FS Literature

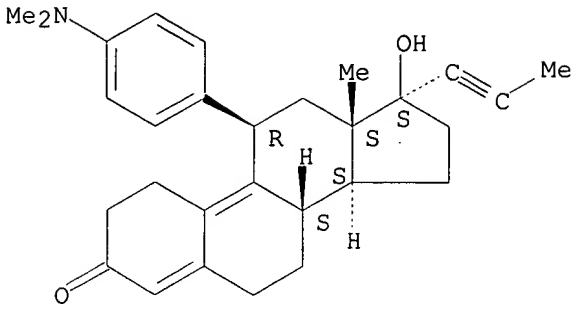
L29 ANSWER 32 OF 36 DRUGU COPYRIGHT 2001 DERWENT INFORMATION LTD
AN 1992-10958 DRUGU T S E
TI The Management of Non-Steroidal Anti-Inflammatory Drug-Induced Gastroduodenal Ulcers.
AU Berge Henegouwen G P van; Smout A J P M
LO Utrecht, Netherlands
SO Scand.J.Gastroenterol. (26, Suppl. 188, 87-91, 1991) 4 Tab. 30 Ref.
CODEN: SJGRA4 ISSN: 0036-5521
AV P.O. Box 85500, 3508, GA Utrecht, The Netherlands.
LA English
DT Journal
FA AB; LA; CT
FS Literature

L29 ANSWER 33 OF 36 CABA COPYRIGHT 2001 CABI
AN 81:1574 CABA
DN 800154773
TI Lordosis induction in the rat by prostaglandin E2 systematically or intracranially in the absence of ovarian hormones
AU Rodriguez-Sierra, J. F.; Komisaruk, B. R.
CS Institute of Animal Behavior, Rutgers State University, 101 Warren Street,
Newark, New Jersey 07102, USA.
SO Prostaglandins, (1978) Vol. 15, No. 3, pp. 513-524. 24 ref.
ISSN: 0090-6980
DT Journal
LA English

L29 ANSWER 34 OF 36 CABA COPYRIGHT 2001 CABI
AN 78:3305 CABA
DN 780135525
TI Facilitation of lordosis in the rat by prostaglandin E2
AU Dudley, C. A.; Moss, R. L.
CS Department of Physiology, Texas University Health Science Center, Southwestern Medical School, Dallas, Texas 75235, USA.
SO Journal of Endocrinology, (1976) Vol. 71, No. 3, pp. 457-458. 8 ref.
ISSN: 0022-0795
DT Journal
LA English

L29 ANSWER 35 OF 36 ADISINSIGHT COPYRIGHT 2001 (ADIS)
ACCESSION NUMBER: 1998:1453 ADISINSIGHT
SOURCE: Adis R&D Insight
DOCUMENT NO: 001657
CHANGE DATE: Jan 31, 2001
GENERIC NAME: Mifepristone
SYNONYM: Mifeprex; RU 38486; RU 486
CHEMICAL NAME: Estra-4,9-dien-3-one, (11beta,17beta)-11-(4-(dimethylamino)phenyl)-17-hydroxy-17-(1-propynyl)-
TRADE NAME: Mifegyne
MOLECULAR FORMULA: C29 H35 N O2
CAS REGISTRY NO.: 84371-65-3
STRUCTURE:

Absolute stereochemistry.



EPHMRA ATC CODE: G2 Other gynaecologicals; L2B Cytostatic Hormone Antagonists
WHO ATC CODE: G03D Progestogens; L02B Hormone Antagonists and Related Agents
HIGHEST DEV. PHASE: Launched

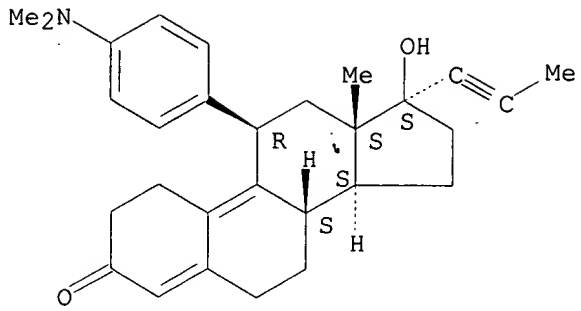
COMPANY INFORMATION
ORIGINATOR: Aventis Pharma (France)
PARENT: Aventis
LICENSEE: Cosan; Exelgyn; Nonindustrial source; Unknown
OTHER: Contragest; Shanghai Hualian Pharmaceutical

OTHER SOURCES: 800671416; 800802900; 800161315; 800238704; 800538779; 800833061; 800690334; 800723592; 800745348; 800813601; 800821978; 800698668
WORD COUNT: 3041

L29 ANSWER 36 OF 36 DRUGUPDATES COPYRIGHT 2001 IMSWORLD

ACCESSION NUMBER: 93:1064 DRUGUPDATES
SOURCE: R&D Focus, (27 Nov 2000)
GENERIC NAME: mifepristone
REFERENCE: INN
LABORATORY NAME: RU 486; RU 4866; RU 38486; R 38486
TRADE NAME: MIFEGYNE; MIFEPREX
CHEMICAL NAME:
(11beta,17beta)-11-[4-(dimethylamino)phenyl]-17-hydroxy-17-(1-propynyl)estra-4,9-dien-3-one
CAS REGISTRY NO.: 84371-65-3
STRUCTURE:

Absolute stereochemistry.



DERIVATIVE(S): 84371-65-3 mifepristone
91934-98-4 N-oxide
105868-38-0 cpd with butyl acetate (1:1)
108179-34-6 fumarate (1:1)
127888-97-5 mixt cont
127888-96-4 mixt with EPA
127888-95-3 mixt with (Z,Z,Z)-8,11,14-eicosatrienoic acid
93781-44-3 (11beta,13alfa,17beta)
91935-24-9 (3E,11beta,17beta), oxime
91935-23-8 (3Z,11beta,17beta), oxime
96346-56-4 (11beta,13alfa,17alfa)
116948-77-7 (11beta,14beta,17alfa)
101978-06-7 (8alfa,11alfa,13alfa,14beta,17alfa)
122742-25-0 replaced by 84371-65-5
83203-42-3 replaced by 84371-65-3

CLASSIFICATION: G2X9 Other Gynecologicals; L2B9 Other Cytostatic Hormone Antagonists; G3A Hormonal Contraceptives, Systemic

HIGHEST DEV. PHASE: Marketed (80)

COMPANY INFORMATION:

Type	Company	Nationality	Region
Originator	Aventis	France	
Licensee	Exelgyn	France	Worldwide outside the USA
Other	Cosan	Switzerland	Switzerland
Other	Danco	United States	United States
Other	Population Council	United States	United States
Assignee	Roussel Uclaf		

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	49.19	282.62

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

STN INTERNATIONAL LOGOFF AT 12:53:23 ON 14 MAY 2001

Connection closed by remote host